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|---|---|---|
| NEWS 1 | | Web Page for STN Seminar Schedule - N. America |
| NEWS 2 | OCT 04 | Precision of EMBASE searching enhanced with new chemical name field |
| NEWS 3 | OCT 06 | Increase your retrieval consistency with new formats or for Taiwanese application numbers in CA/CAplus. |
| NEWS 4 | OCT 21 | CA/CAplus kind code changes for Chinese patents increase consistency, save time |
| NEWS 5 | OCT 22 | New version of STN Viewer preserves custom highlighting of terms when patent documents are saved in .rtf format |
| NEWS 6 | OCT 28 | INPADOCDB/INPAFAMDB: Enhancements to the US national patent classification. |
| NEWS 7 | NOV 03 | New format for Korean patent application numbers in CA/CAplus increases consistency, saves time. |
| NEWS 8 | NOV 04 | Selected STN databases scheduled for removal on December 31, 2010 |
| NEWS 9 | NOV 18 | PROUSDDR and SYNTHLINE Scheduled for Removal December 31, 2010 by Request of Prous Science |
| NEWS 10 | NOV 22 | Higher System Limits Increase the Power of STN Substance-Based Searching |
| NEWS 11 | NOV 24 | Search an additional 46,850 records with MEDLINE backfile extension to 1946 |
| NEWS 12 | DEC 14 | New PNK Field Allows More Precise Crossover among STN Patent Databases |
| NEWS 13 | DEC 18 | ReaxysFile available on STN |
| NEWS 14 | DEC 21 | CAS Learning Solutions -- a new online training experience |
| NEWS 15 | DEC 22 | Value-Added Indexing Improves Access to World Traditional Medicine Patents in CAplus |
| NEWS 16 | JAN 24 | The new and enhanced DPCI file on STN has been released |
| NEWS 17 | JAN 26 | Improved Timeliness of CAS Indexing Adds Value to USPATFULL and USPAT2 Chemistry Patents |
| NEWS 18 | JAN 26 | Updated MeSH vocabulary, new structured abstracts, and other enhancements improve searching in STN reload of MEDLINE |
| NEWS 19 | JAN 28 | CABA will be updated weekly |
| NEWS 20 | FEB 23 | PCTFULL file on STN completely reloaded |
| NEWS 21 | FEB 23 | STN AnaVist Test Projects Now Available for Qualified Customers |
| NEWS 22 | FEB 25 | LPCI will be replaced by LDPCI |
| NEWS 23 | MAR 07 | Pricing for SELECTing Patent, Application, and Priority Numbers in the USPAT and IFI Database Families is Now Consistent with Similar Patent Databases on STN |

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FILE 'HOME' ENTERED AT 09:39:24 ON 31 MAR 2011

=> file caplus biosis
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.23       0.23

FILE 'CAPLUS' ENTERED AT 09:39:40 ON 31 MAR 2011
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FILE 'BIOSIS' ENTERED AT 09:39:40 ON 31 MAR 2011
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=> OX40 (L) transcriptional (w) factor
L1            0 OX40 (L) TRANSCRIPTIONAL (W) FACTOR

=> OX40 (L) NFkepaB
L2            0 OX40 (L) NFKEPAB

=> NFkB and OX40
L3            1 NFKB AND OX40

=> (Ap-1) and OX40
L4           10 (AP-1) AND OX40

=> D L3 1BIB ABS

L3   ANSWER 1 OF 1  BIOSIS  COPYRIGHT (c) 2011 The Thomson Corporation  on STN
 Full Text  Citing References
ACCESSION NUMBER:    2005:477815  BIOSIS
DOCUMENT NUMBER:     PREV200510269719
TITLE:               Three-module signaling endo-domain artifical T-cell
                     receptor which transmits CD28, OX40 and CD3-xi signals
                     enhances IL-2 release and proliferative response in
                     transduced primary T-cells.
AUTHOR(S):           Pule, Martin A. [Reprint Author]; Straathof, Karin C.;
                     Dotti, Gianpietro; Heslop, Helen E.; Rooney, Cliona M.;
                     Brenner, Malcolm K.
CORPORATE SOURCE:    Baylor Coll Med, Ctr Cell and Gene Therapy, Houston, TX
                     77030 USA
SOURCE:              Blood, (NOV 16 2004) Vol. 104, No. 11, Part 1, pp. 484A.
                     Meeting Info.: 46th Annual Meeting of the
                     American-Society-of-Hematology. San Diego, CA, USA.
                     December 04 -07, 2004. Amer Soc Hematol.
                     CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE:       Conference; (Meeting)
                     Conference; (Meeting Poster)
LANGUAGE:            English
ENTRY DATE:          Entered STN: 16 Nov 2005
                     Last Updated on STN: 16 Nov 2005
AB   Artificial T-cell receptors (TCR) are generated by connecting an antigen
     recognizing ectodomain to a signal transducing endodomain.  Most
     frequently the variable chains of Immunoglobulin molecules expressed as a
     single chain (ScFv) areutilized as ectodomains and the intracellular
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portion of CD3-zeta is used as endodomain. When expressed by primary T-cells these molecules can redirect thecellular immune response to almost any surface target molecule for which a monoclonal antibody can be made. However, clinical studies with these chimeric T-cells have been disappointing, with no clear clinical benefit, and only minimal in vivo persistence of infused T-cells. Transmitted CD3-zeta signal is onlysufficient to activate cell-killing and Inteferon-gamma release but fails to induce IL-2 release or proliferation. Full T cell activation requires co-stimulatory signals that are rarely provided by the tumor cells and therefore may need to be incorporated in the endodomain of the artificial TCR. Indeed, inclusion of a CD28 signaling component resulted in IL-2 release and limited Proliferation, but T cell activation appears still incomplete. **OX40** is a TNFR family molecule expressed by activated T-cells. It transmits a potent and prolonged activation signal and has been found to be an important molecule for maintaining a prolonged immunological response e.g. in chronic inflammation. We held thehypothesis that an artificial TCR providing 3 signals - CD3-zeta, CD28 and **OX40** in cis would result in more potent activation and more prolonged proliferation. We generated and compared a number of constructs based on GD2 recognizingscFv 14g2a: 14g2a-zeta, 14g2a-CD28-zeta, 14g2a-**OX40**-zeta, 14g2a-CD28-**OX40**-zeta. We first co-immunoprecipitated TRAF-2 with **OX40** containing constructs. Thisdemonstrated that the **OX40** binding site was unaffected by fusion with other proteins. Incorporating 3 signals - CD3-zeta, CD28 and **OX40** in cis from a single endodomain of an artificial TCR recruited a 10 fold higher level of **NFkB** quantified by Luciferase-reporter than two signals (14g2a.CD28-zeta) and over 50 fold higher than a single signal (14g2a.)T-cells transduced with all of these constructs were capable of lysing GD2+ neuroblastoma cells. Only limited expansion (1.6 fold. range 0.9-3) was induced upon stimulation with tumor cells in T cells transduced with 14g2a.**OX40**ζ Adding a CD28 domain resulted in a 5.2 fold (range: 1.6-7.2) expansion within 7 days but this proliferation could not be maintained. In contrast, 14g2a.CD28.**OX40**ζ transduced T cells expanded 10.7 fold (range: 4-17) within 7 days and continued to proliferate with weekly stimulations with tumor cells, even in the absence of exogenous IL-2. Thisincreased proliferation of 14g2a.CD28.**OX40**ζ transduced T cells was accompanied by a > 10-fold increase in IL-2 and 5-fold increase in TNF-a secretion as compared to the 14g2a.CD28-zeta construct. Sustained proliferation was accompanied by persisting function - T-cells transduced with 14g2a.CD28-**OX40**-zeta were still capable of killing GD2+ targets after 35 days of culture. These improved functional characteristics should favor the overall utility of chimeric T-cells.

=> D L4 TERM ABS 1-10

L4 ANSWER 1 OF 10 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Other References

| | |
|---|---|
| ACCESSION NUMBER: | 2007:221241 CAPLUS |
| DOCUMENT NUMBER: | 146:399193 |
| TITLE: | Human T cell leukemia virus type 1 Tax-induced signals in cell survival, proliferation, and transformation |
| AUTHOR(S): | Silbermann, Katrin; Grassmann, Ralph |
| CORPORATE SOURCE: | Institut fuer Klinische und Molekulare Virologie, Friedrich-Alexander Universitaet Erlangen-Nuernberg, Erlangen, Germany |
| SOURCE: | Signal Transduction (2007), 7(1), 34-52 CODEN: STIRCI; ISSN: 1615-4053 |
| PUBLISHER: | Wiley-VCH Verlag GmbH & Co. KGaA |

DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Human T cell leukemia virus type 1 (HTLV-1), a delta-retrovirus, causes an aggressive malignancy of T lymphocytes called adult T cell leukemia/lymphoma and stimulates permanent cell growth in culture. The virus encodes a nonstructural regulatory protein, Tax, which is both transforming in cell culture and oncogenic in vivo. This multifunctional protein controls viral transcription and in multiple ways interferes with cellular control of survival, proliferation, and genomic stability. Tax, by activation of NF-κB, **AP-1**, and other transcriptional pathways, enhances expression of cellular genes encoding cytokines (e.g. IL-13, IL-15), cytokine receptors (e.g. IL-2Rα), and antiapoptotic factors (Hiap-1, Bcl-xL, **OX40**), leading to altered signal transduction (e.g. Jak/Stat, PI3K, Caspase 3/7). Cellular proliferation is stimulated by direct targeting of the cell cycle kinase (Cdk4, Cdk6) holoenzymes, repression of Cdk inhibitors, and the functional inactivation of the tumor suppressor p53. Finally, Tax, by promoting genomic instability through interference with DNA-damage signaling, checkpoint control (G2/M, mitotic spindle assembly), chromosome segregation, and cellular DNA repair pathways could contribute to malignant conversion of infected cells.
OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)
REFERENCE COUNT: 258 THERE ARE 258 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Ref Referen ces

ACCESSION NUMBER: 2006:694614 CAPLUS
DOCUMENT NUMBER: 145:122838
TITLE: PKC-θ-Deficient Mice Are Protected from Th1-Dependent Antigen-Induced Arthritis
AUTHOR(S): Healy, Aileen M.; Izmailova, Elena; Fitzgerald, Michael; Walker, Russell; Hattersley, Maureen; Silva, Matthew; Siebert, Elizabeth; Terkelsen, Jennifer; Picarella, Dominic; Pickard, Michael D.; LeClair, Brett; Chandra, Sudeep; Jaffee, Bruce
CORPORATE SOURCE: Inflammation Department and Imaging Sciences, Millennium Pharmaceuticals, Cambridge, MA, 02139, USA
SOURCE: Journal of Immunology (2006), 177(3), 1886-1893 CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English
AB T cell effector functions contribute to the pathogenesis of rheumatoid arthritis. PKC-θ transduces the signal from the TCR through activation of transcription factors NF-κB, **AP-1**, and NFAT. The authors examd. the effects of PKC-θ deficiency on two Th1-dependent models of Ag-induced arthritis and found that PKC-θ-deficient mice develop disease, but at a diminished severity compared with wild-type mice. In the methylated BSA model, cellular infiltrates and articular cartilage damage were mild in the PKC-θ-deficient mice as compared with wild-type mice. Quantitation of histopathol. reveals 63% and 77% redn. in overall joint destruction in two independent expts. In the type II collagen-induced arthritis model, the authors obsd. a redn. in clin. scores in 3 independent expts. and diminished joint pathol. in PKC-θ-deficient compared with wild-type littermates. Microcomputerized tomog. imaging revealed that PKC-θ deficiency also

protects from bone destruction. PKC-θ-deficient CD4+ T cells show an impaired proliferative response, decreased intracellular levels of the cytokines IFN-γ, IL-2, and IL-4, and diminished cell surface expression of the activation markers CD25, CD69, and CD134/**OX40** on memory T cells. The authors demonstrate decreased T-bet expression and reduced IgG1 and IgG2a anti-collagen II Ab levels in PKC-θ-deficient mice. Thus, PKC-θ deficiency results in an attenuated response to Ag-induced arthritis, which is likely mediated by the reduced T cell proliferation, Th1/Th2 cell differentiation and T cell activation before and during disease peak.

OS.CITING REF COUNT: 41 THERE ARE 41 CAPLUS RECORDS THAT CITE THIS RECORD (41 CITINGS)

REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 3 OF 10 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | More References

ACCESSION NUMBER: 2006:352467 CAPLUS

DOCUMENT NUMBER: 144:430984

TITLE: STAT3 and NF-κB Signal Pathway Is Required for IL-23-Mediated IL-17 Production in Spontaneous Arthritis Animal Model IL-1 Receptor Antagonist-Deficient Mice

AUTHOR(S): Cho, Mi-La; Kang, Jung-Won; Moon, Young-Mee; Nam, Hyo-Jung; Jhun, Joo-Yeon; Heo, Seong-Beom; Jin, Hyun-Tak; Min, So-Youn; Ju, Ji-Hyeon; Park, Kyung-Su; Cho, Young-Gyu; Yoon, Chong-Hyeon; Park, Sung-Hwan; Sung, Young-Chul; Kim, Ho-Youn

CORPORATE SOURCE: The Rheumatism Research Center, Catholic Research Institute of Medical Science, Catholic University of Korea, Seoul, 137-040, S. Korea

SOURCE: Journal of Immunology (2006), 176(9), 5652-5661 CODEN: JOIMA3; ISSN: 0022-1767

PUBLISHER: American Association of Immunologists

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Interleukin 23 (IL-23) is a heterodimeric cytokine composed of a p19 subunit and the p40 subunit of IL-12. IL-23 has proinflammatory activity, inducing IL-17 secretion by activated CD4+ T cells and stimulating the proliferation of memory CD4+ T cells. The authors investigated the pathogenic role of IL-23 in CD4+ T cells in mice lacking the IL-1R antagonist (IL-1Ra-/-), an animal model of spontaneous arthritis. IL-23 was strongly expressed in the inflamed joints of IL-1Ra-/- mice. Recombinant adenovirus expressing mouse IL-23 (rAd/mIL-23) accelerated this joint inflammation and joint destruction. IL-1β further increased the prodn. of IL-23, which induced IL-17 prodn. and **OX40** expression in splenic CD4+ T cells of IL-1Ra-/- mice. Blocking IL-23 with anti-p19 Ab abolished the IL-17 prodn. induced by IL-1 in splenocyte cultures. The process of IL-23-induced IL-17 prodn. in CD4+ T cells was mediated via the activation of Jak2, PI3K/Akt, STAT3, and NF-κB, whereas p38 MAPK and **AP-1** did not participate in the process. The authors' data suggest that IL-23 is a link between IL-1 and IL-17. IL-23 seems to be a central proinflammatory cytokine in the pathogenesis of this IL-1Ra-/- model of spontaneous arthritis. Its intracellular signaling pathway could be a useful therapeutic target in the treatment of autoimmune arthritis.

OS.CITING REF COUNT: 77 THERE ARE 77 CAPLUS RECORDS THAT CITE THIS RECORD (77 CITINGS)

REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 4 OF 10 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Cited References

ACCESSION NUMBER: 2005:729611 CAPLUS
DOCUMENT NUMBER: 143:206465
TITLE: Therapeutic and carrier molecules
INVENTOR(S): Ferrante, Antonio; Rathjen, Deborah Ann
PATENT ASSIGNEE(S): Peplin Biolipids Pty Ltd, Australia
SOURCE: PCT Int. Appl., 180 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2005073164 | A1 | 20050811 | WO 2005-AU98 | 20050128 |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW | | | | |
| RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| AU 2005209331 | A1 | 20050811 | AU 2005-209331 | 20050128 |
| CA 2554735 | A1 | 20050811 | CA 2005-2554735 | 20050128 |
| EP 1718602 | A1 | 20061108 | EP 2005-700130 | 20050128 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK, IS | | | | |
| CN 1934072 | A | 20070321 | CN 2005-80008891 | 20050128 |
| BR 2005007236 | A | 20070626 | BR 2005-7236 | 20050128 |
| JP 2007522118 | T | 20070809 | JP 2006-549788 | 20050128 |
| US 20090215895 | A1 | 20090827 | US 2009-588094 | 20090507 |
| PRIORITY APPLN. INFO.: | | | US 2004-540604P P | 20040130 |
| | | | WO 2005-AU98 W | 20050128 |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OTHER SOURCE(S): MARPAT 143:206465

AB The present invention relates generally to compds. comprising a hydrocarbon chain portion and more particular to compds. comprising chem. derivatizations of the hydrocarbon chain which are useful therapeutic and prophylactic mols. The present invention further provides compds. where the hydrocarbon chain portion is a carrier mol. for functional groups, moieties or agents. The present invention can include naturally including polyunsatd. fatty acids as well as synthetic, modified or derivatized polyunsatd. fatty acids. Furthermore. these polyunsatd. fatty acids can be conjugated to amino acids, peptides or proteins. The compds. of the present invention are particularly useful in the treatment and prophylaxis of a range of conditions including cancers, protein kinase c(PKC)- or NFκB-related- or -assocd. conditions, cardiovascular conditions, pain, inflammatory conditions, vascular or immunol. conditions such as diabetes, neurol. conditions and infection by a range of viruses or prokaryotic or eukaryotic organisms. The present invention further provides pharmaceutical compns. and methods of medical treatment.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)
REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 5 OF 10 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2004:1156439 CAPLUS
DOCUMENT NUMBER: 142:73408
TITLE: DNA vaccines comprising immunomodulatory proteins and antigen from pathogens
INVENTOR(S): Weiner, David B.; Muthumani, Karuppiah; Kutzler, Michele; Choo, Andrew K.; Chattergoon, Michael A.
PATENT ASSIGNEE(S): The Trustees of the University of Pennsylvania, USA
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2004112706 | A2 | 20041229 | WO 2004-US19028 | 20040614 |
| WO 2004112706 | A3 | 20050414 | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW | | | | |
| RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| AU 2004249191 | A1 | 20041229 | AU 2004-249191 | 20040614 |
| AU 2004249191 | B2 | 20110106 | | |
| CA 2529051 | A1 | 20041229 | CA 2004-2529051 | 20040614 |
| EP 1633372 | A2 | 20060315 | EP 2004-755303 | 20040614 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK | | | | |
| JP 2007502868 | T | 20070215 | JP 2006-533794 | 20040614 |
| US 20070104686 | A1 | 20070510 | US 2004-560653 | 20040614 |
| PRIORITY APPLN. INFO.: | | | US 2003-478187P P | 20030613 |
| | | | US 2003-478230P P | 20030613 |
| | | | US 2003-478250P P | 20030613 |
| | | | WO 2004-US19028 W | 20040614 |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The authors disclose the use of recombinant vaccines and live attenuated pathogens comprising one or more isolated nucleic acid mols. that encode an immunogen in combination with an isolated nucleic acid mol. that encodes an immunomodulator protein selected from the group consisting of: Fos, c-jun, Sp-1, AP-1, AP-2, p38, p65Rel, MyD88, IRAK, TRAF6, IκB, inactive NIK, SAP kinase, SAP-1, JNK, interferon response genes, NF-κB, Bax, TRAIL, TRAIL receptors, DcR5, TRAIL-R3, TRAIL-R4, RANK, RANK ligand, **Ox40**, **Ox40** ligand, NKG2D, MICA, MICB, NKG2A, NKG2B, NKG2C, NKG2E, NKG2F, TAP1, TAP2 and functional fragments thereof.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD

(3 CITINGS)
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 6 OF 10 BIOSIS COPYRIGHT (c) 2011 The Thomson Corporation on STN

Full Text | Citing References

ACCESSION NUMBER: 2007:109136 BIOSIS
DOCUMENT NUMBER: PREV200700109662
TITLE: Il-23-mediated Il-17 production via stat3 and Nf-kb signal pathway in spontaneous arthritis animal model, Il-1 receptor antagonist-deficient mice.
AUTHOR(S): Seo, Soo-Hong [Reprint Author]; Yoon, Chong-Hyeon; Ju, Ji-Hyeon; Kwok, Seung-Hwan; Park, Sung-Hwan; Cho, Chul-Soo; Kim, Ho-Youn; Cho, Mi-La
CORPORATE SOURCE: Catholic Univ Korea, Kangnam St Marys Hosp, Seoul, South Korea
SOURCE: Arthritis & Rheumatism, (SEP 2006) Vol. 54, No. 9, Suppl. S, pp. S593-S594.
Meeting Info.: 70th Annual Scientific Meeting of the American-College-of-Rheumatology/41st Annual Scientific Meeting of the Association-of-Rheumatology-Health-Professionals. Washington, DC, USA. November 10 -15, 2006.
Amer Coll Rheumatol; Assoc Rheumatol Hlth Profess.
CODEN: ARHEAW. ISSN: 0004-3591.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Feb 2007
Last Updated on STN: 14 Feb 2007

L4 ANSWER 7 OF 10 BIOSIS COPYRIGHT (c) 2011 The Thomson Corporation on STN

Full Text | Citing References

ACCESSION NUMBER: 2006:408842 BIOSIS
DOCUMENT NUMBER: PREV200600406241
TITLE: PKC-theta-deficient mice are protected from Th1-dependent antigen-induced arthritis.
AUTHOR(S): Healy, Aileen M. [Reprint Author]; Izmailova, Elena; Fitzgerald, Michael; Walker, Russell; Hattersley, Maureen; Silva, Matthew; Siebert, Elizabeth; Terkelsen, Jennifer; Picarella, Dominic; Pickard, Michael D.; LeClair, Brett; Chandra, Sudeep; Jaffee, Bruce
CORPORATE SOURCE: Momenta Pharmaceut, 675 W Kendall St, Cambridge, MA 02142 USA
ahealy@momentapharma.com
SOURCE: Journal of Immunology, (AUG 1 2006) Vol. 177, No. 3, pp. 1886-1893.
CODEN: JOIMA3. ISSN: 0022-1767.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 17 Aug 2006
Last Updated on STN: 17 Aug 2006

AB T cell effector functions contribute to the pathogenesis of rheumatoid arthritis. PKC-theta transduces the signal from the TCR through activation of transcription factors NF-kappa B, **AP-1**, and NFAT. We examined the effects of PKC-theta deficiency on two Th1-dependent models of Ag-induced arthritis and found that PKC-theta-deficient mice-develop disease, but at a significantly diminished severity compared with wild-type mice. In the methylated BSA model, cellular infiltrates and

articular cartilage damage were mild in the PKC-theta-deficient mice as compared with wild-type mice. Quantitation of histopathology reveals 63 and 77% reduction in overall joint destruction in two independent experiments. In the type II collagen-induced arthritis model, we observed a significant reduction in clinical scores ($p < 0.01$) in three independent experiments and diminished joint pathology ($p < 0.005$).in PKC-theta-deficient compared with wild-type littermates. Microcomputerized tomographic imaging revealed that PKC-theta deficiency also protects from bone destruction. PKC-theta-deficient CD4(+) T cells show an impaired proliferative response, decreased intracellular levels of the cytokines IFN-gamma, IL-2, and IL-4, and significantly diminished cell surface expression of the activation markers CD25, CD69, and CD134/**OX40** on memory T cells. We demonstrate decreased T-bet expression and significantly reduced IgG1 and IgG2a anti-collagen II Ab levels in PKC-theta-deficient mice. Collectively, our results demonstrate that PKC-theta deficiency results in an attenuated response to Ag-induced arthritis, which is likely mediated by the reduced T cell proliferation, Th1/Th2 cell differentiation and T cell activation before and during disease peak.

L4 ANSWER 8 OF 10 BIOSIS COPYRIGHT (c) 2011 The Thomson Corporation on STN

Full Text | Citing References

ACCESSION NUMBER: 2006:399161 BIOSIS
DOCUMENT NUMBER: PREV200600394535
TITLE: STAT3 and NF-kappa B signal pathway is required for IL-23-mediated IL-17 production in spontaneous arthritis animal model IL-1 receptor antagonist-deficient mice.
AUTHOR(S): Cho, Mi-La; Kang, Jung-Won; Moon, Young-Mee; Nam, Hyo-Jung; Jhun, Joo-Yeon; Heo, Seong-Beom; Jin, Hyun-Tak; Min, So-Youn; Ju, Ji-Hyeon; Park, Kyung-Su; Cho, Young-Gyu; Yoon, Chong-Hyeon; Park, Sung-Hwan; Sung, Young-Chul; Kim, Ho-Youn [Reprint Author]
CORPORATE SOURCE: Catholic Univ Korea, Catholic Res Inst Med Sci, Rheumatism Res Ctr, 505 Banpo Dong, Seoul, South Korea
ho@catholic.ac.kr
SOURCE: Journal of Immunology, (MAY 1 2006) Vol. 176, No. 9, pp. 5652-5661.
CODEN: JOIMA3. ISSN: 0022-1767.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Aug 2006
Last Updated on STN: 9 Aug 2006

AB IL-23 is a heterodimeric cytokine composed of a p19 subunit and the p40 subunit of IL-12. IL-23 has proinflammatory activity, inducing IL-17 secretion from activated CD4(+) T cells and stimulating the proliferation of memory CD4(+) T cells. We investigated the pathogenic role of IL-23 in CD4(+) T cells in mice lacking the IL-1R antagonist (1L-1Ra(-/-)), an animal model of spontaneous arthritis. IL-23 was strongly expressed in the inflamed joints of IL-1Ra(-/-) mice. Recombinant adenovirus expressing mouse IL-23 (rAd/mIL-23) significantly accelerated this joint inflammation and joint destruction. IL-1 beta further increased the production of IL-23, which induced IL-17 production and **OX40** expression in splenic CD4(+) T cells of IL-1Ra(-/-) mice. Blocking IL-23 with anti-p19 Ab abolished the IL-17 production induced by IL-1 in splenocyte cultures. The process of IL-23-induced IL-17 production in CD4(+) T cells was mediated via the activation of Jak2, PI3K/Akt, STAT3, and NF-kappa B, whereas p38 MAPK and **AP-1** did not participate in the process. Our data suggest that IL-23 is a link between IL-1 and IL-17. IL-23 seems to be a central proinflammatory cytokine in the pathogenesis of this

IL-1Ra(-/-) model of spontaneous arthritis. Its intracellular signaling pathway could be useful therapeutic targets in the treatment of autoimmune arthritis.

L4 ANSWER 9 OF 10 BIOSIS COPYRIGHT (c) 2011 The Thomson Corporation on STN

Full Text | More References

ACCESSION NUMBER: 2005:534958 BIOSIS
DOCUMENT NUMBER: PREV200510320461
TITLE: Dual alpha 4-integrin antagonists inhibit T cell activation and IL-2 production following specific costimulation with anti-CD3 and VCAM-1.
AUTHOR(S): Cohn, Ronald Gary [Reprint Author]; Lau, Bonnie; Levin, Anita; Sidduri, Achyutharao; Tilley, Jefferson; Renzetti, Louis; Fuentes, Maria
CORPORATE SOURCE: Roche Palo Alto LLC, Palo Alto, CA 94304 USA
SOURCE: FASEB Journal, (MAR 7 2005) Vol. 19, No. 5, Suppl. S, Part 2, pp. A1449.
Meeting Info.: Experimental Biology 2005 Meeting/35th International Congress of Physiological Sciences. San Diego, CA, USA. March 31 -April 06, 2005. Amer Assoc Anatomists; Amer Assoc Immunologists; Amer Physiol Soc; Amer Soc Biochem & Mol Biol; Amer Soc Investigat Pathol; Amer Soc Nutr Sci; Amer Soc Pharmacol & Expt Therapeut; Int Union Physiol Sci.
CODEN: FAJOEC. ISSN: 0892-6638.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Dec 2005
Last Updated on STN: 1 Dec 2005

AB Binding of the integrins alpha 4 beta 1 (VLA-4) and alpha 4 beta 7 to their counter ligands, VCAM-1 and MadCAM-1, are critical interactions leading to migration of lymphocytes into tissues. Additionally, co-stimulation of T cells withrecombinant VCAM-1 has been shown to enhance induction of transcription factors **AP-1**, NF-AT, and NF-kappa B, leading- to increased secretion Of Multiple inflammatory cytokines which occur in chronic inflammatory diseases such as asthma, rheumatoid arthritis, multiple sclerosis, and inflammatory bowel disease. Most current therapies available to treat these diseases have undesirable sideeffects with long-term usage. Inhibitory anti-integrin monoclonal antibodies are proving effective treatments for some chronic inflammatory diseases, but their administration to patients and cost make development of small molecule integrin-antagonists desirable. Here we report that co-stimulation of purified Tcells with anti-CD3 and VCAM-1 increased production of IL2 and induced expression of **OX40** (CD134) and CD69. This costimulation regimen induced these markers at levels higher than costimulation with anti-CD3 alone or in combination with anti-CD28, demonstrating the integrin specificity of the co-stimulatory signal. These responses were attenuated by the dual alpha 4-integrin antagonists RO0270608, R00504183, and R00505291. Thus, in addition to blocking T cell trafficking, dual a4-integrin antagonists may promote anti-inflammatory activity by directly modulating T cell function, i.e., blockade of T cell proliferation signaling through **OX40**.

L4 ANSWER 10 OF 10 BIOSIS COPYRIGHT (c) 2011 The Thomson Corporation on

Full Text | More References

STN
ACCESSION NUMBER: 2005:534957 BIOSIS

DOCUMENT NUMBER: PREV200510320460
TITLE: Effect of Immunotherapy with ISS-ODN and allergen in animal model of timothy allergy.
AUTHOR(S): Hill, Brandon D. [Reprint Author]; Jaechun, Lee; Zhou, Bin; Yoo, T. J.
CORPORATE SOURCE: Univ Tennessee, Dept Allergy and Immunol, Memphis, TN 38163 USA
SOURCE: FASEB Journal, (MAR 7 2005) Vol. 19, No. 5, Suppl. S, Part 2, pp. A1449.
Meeting Info.: Experimental Biology 2005 Meeting/35th International Congress of Physiological Sciences. San Diego, CA, USA. March 31 -April 06, 2005. Amer Assoc Anatomists; Amer Assoc Immunologists; Amer Physiol Soc; Amer Soc Biochem & Mol Biol; Amer Soc Investigat Pathol; Amer Soc Nutr Sci; Amer Soc Pharmacol & Expt Therapeut; Int Union Physiol Sci.
CODEN: FAJOEC. ISSN: 0892-6638.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Dec 2005
Last Updated on STN: 1 Dec 2005

AB Binding of the integrins alpha 4 beta 1 (VLA-4) and alpha 4 beta 7 to their counter ligands, VCAM-1 and MadCAM-1, are critical interactions leading to migration of lymphocytes into tissues. Additionally, co-stimulation of T cells withrecombinant VCAM-1 has been shown to enhance induction of transcription factors **AP-1**, NF-AT, and NF-kappa B, leading to increased secretion Of Multiple inflammatory cytokines which occur in chronic inflammatory diseases such as asthma, rheumatoid arthritis, multiple sclerosis, and inflammatory bowel disease. Most current therapies available to treat these diseases have undesirable side effects with long-term usage. Inhibitory anti-integrin monoclonal antibodies are proving effective treatments for some chronic inflammatory diseases, but their administration to patients and cost make development of small molecule integrin-antagonists desirable. Here we report that co-stimulation of purified T cells with anti-CD3 and VCAM-1 increased production of IL2 and induced expression of **OX40** (CD134) and CD69. This costimulation regimen induced these markersat levels higher than costimulation with anti-CD3 alone or in combination with anti-CD28, demonstrating the integrin specificity of the co-stimulatory signal. These responses were attenuated by the dual a4-integrin antagonists RO0270608, R00504183, and R00505291. Thus, in addition to blocking T cell trafficking, dual a4-integrin antagonists may promote anti-inflammatory activity by directly modulating T cell function, i.e., blockade of T cell proliferation signaling through **OX40**.

```
=> MAP (w) p38
L5          20 MAP (W) P38

=> OX40 and L5
L6           0 OX40 AND L5

=> Fos and OX40
L7           7 FOS AND OX40

=> Jun and OX40
L8          15 JUN AND OX40

=> L8 not OX40L
```

```
L9           11 L8 NOT OX40L

=> (Ap-2) and OX40
L10           1 (AP-2) AND OX40

=> p38 and OX40
L11           9 P38 AND OX40

=> L11 not OX40L
L12           5 L11 NOT OX40L

=> p65Rel and OX40
L13           1 P65REL AND OX40

=> MyD88 and OX40
L14           8 MYD88 AND OX40

=> L14 not OX40L
L15           6 L14 NOT OX40L

=> IRAK and OX40
L16           3 IRAK AND OX40

=> L16 not OX40L
L17           2 L16 NOT OX40L

=> TRAF6 and OX40
L18           7 TRAF6 AND OX40

=> L18 not OX40L
L19           6 L18 NOT OX40L

=> (SAP-1) and OX40
L20           1 (SAP-1) AND OX40

=> Bax and OX40
L21          10 BAX AND OX40

=> L21 not OX40L
L22           6 L21 NOT OX40L

=> JNK and OX40
L23           8 JNK AND OX40

=> L23 not OX40L
L24           3 L23 NOT OX40L

=> Ikb and OX40
L25           2 IKB AND OX40

=> L25 not OX40L
L26           2 L25 NOT OX40L

=> RANK and OX40
AND IS NOT VALID HERE
The term is either unrecognized or invalid.

=> NKG2D and OX40
L27           6 NKG2D AND OX40
```

```
=> L27 not OX40L
L28             6 L27 NOT OX40L

=> MICA and OX40
L29             2 MICA AND OX40

=> L29 not OX40L
L30             2 L29 NOT OX40L

=> NKG2A and OX40
L31             4 NKG2A AND OX40

=> L31 not OX40L
L32             4 L31 NOT OX40L

=> TAP1 and OX40
L33             1 TAP1 AND OX40

=> TAP2 and OX40
L34             1 TAP2 AND OX40

=> NKG2$1
SYSTEM LIMITS EXCEEDED - SEARCH ENDED
The search profile you entered was too complex or gave too many
answers.  Simplify or subdivide the query and try again.  If you have
exceeded the answer limit, enter DELETE HISTORY at an arrow prompt
(=>) to remove all previous answers sets and begin at L1.  Use the
SAVE command to store any important profiles or answer sets before
using DELETE HISTORY.

=> L7 not OX40L
L35             6 L7 NOT OX40L

=> D L34 TRIB ABS

L34  ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2011 ACS on STN
```

Full Text | Citing References

| | |
|---|---|
| ACCESSION NUMBER: | 2004:1156439 CAPLUS |
| DOCUMENT NUMBER: | 142:73408 |
| TITLE: | DNA vaccines comprising immunomodulatory proteins and antigen from pathogens |
| INVENTOR(S): | Weiner, David B.; Muthumani, Karuppiah; Kutzler, Michele; Choo, Andrew K.; Chattergoon, Michael A. |
| PATENT ASSIGNEE(S): | The Trustees of the University of Pennsylvania, USA |
| SOURCE: | PCT Int. Appl., 47 pp.<br>CODEN: PIXXD2 |
| DOCUMENT TYPE: | Patent |
| LANGUAGE: | English |
| FAMILY ACC. NUM. COUNT: | 1 |
| PATENT INFORMATION: | |

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2004112706 | A2 | 20041229 | WO 2004-US19028 | 20040614 |
| WO 2004112706 | A3 | 20050414 | | |

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,

NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
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EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE,
SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
SN, TD, TG

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|---|---|---|---|---|
| AU 2004249191 | A1 | 20041229 | AU 2004-249191 | 20040614 |
| AU 2004249191 | B2 | 20110106 | | |
| CA 2529051 | A1 | 20041229 | CA 2004-2529051 | 20040614 |
| EP 1633372 | A2 | 20060315 | EP 2004-755303 | 20040614 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK | | | | |
| JP 2007502868 | T | 20070215 | JP 2006-533794 | 20040614 |
| US 20070104686 | A1 | 20070510 | US 2004-560653 | 20040614 |
| PRIORITY APPLN. INFO.: | | | US 2003-478187P | P 20030613 |
| | | | US 2003-478230P | P 20030613 |
| | | | US 2003-478250P | P 20030613 |
| | | | WO 2004-US19028 | W 20040614 |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The authors disclose the use of recombinant vaccines and live attenuated pathogens comprising one or more isolated nucleic acid mols. that encode an immunogen in combination with an isolated nucleic acid mol. that encodes an immunomodulator protein selected from the group consisting of: Fos, c-jun, Sp-1, AP-1, AP-2, p38, p65Rel, MyD88, IRAK, TRAF6, IκB, inactive NIK, SAP kinase, SAP-1, JNK, interferon response genes, NF-κB, Bax, TRAIL, TRAIL receptors, DcR5, TRAIL-R3, TRAIL-R4, RANK, RANK ligand, **Ox40**, **Ox40** ligand, NKG2D, MICA, MICB, NKG2A, NKG2B, NKG2C, NKG2E, NKG2F, TAP1, **TAP2** and functional fragments thereof.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> D L20 IBIB ABS

L20 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | RHG References

ACCESSION NUMBER: 2004:1156439 CAPLUS
DOCUMENT NUMBER: 142:73408
TITLE: DNA vaccines comprising immunomodulatory proteins and antigen from pathogens
INVENTOR(S): Weiner, David B.; Muthumani, Karuppiah; Kutzler, Michele; Choo, Andrew K.; Chattergoon, Michael A.
PATENT ASSIGNEE(S): The Trustees of the University of Pennsylvania, USA
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2004112706 | A2 | 20041229 | WO 2004-US19028 | 20040614 |
| WO 2004112706 | A3 | 20050414 | | |
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LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE,
SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
SN, TD, TG

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|---|---|---|---|---|---|
| AU 2004249191 | A1 | 20041229 | AU 2004-249191 | | 20040614 |
| AU 2004249191 | B2 | 20110106 | | | |
| CA 2529051 | A1 | 20041229 | CA 2004-2529051 | | 20040614 |
| EP 1633372 | A2 | 20060315 | EP 2004-755303 | | 20040614 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK | | | | | |
| JP 2007502868 | T | 20070215 | JP 2006-533794 | | 20040614 |
| US 20070104686 | A1 | 20070510 | US 2004-560653 | | 20040614 |
| PRIORITY APPLN. INFO.: | | | US 2003-478187P | P | 20030613 |
| | | | US 2003-478230P | P | 20030613 |
| | | | US 2003-478250P | P | 20030613 |
| | | | WO 2004-US19028 | W | 20040614 |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The authors disclose the use of recombinant vaccines and live attenuated pathogens comprising one or more isolated nucleic acid mols. that encode an immunogen in combination with an isolated nucleic acid mol. that encodes an immunomodulator protein selected from the group consisting of: Fos, c-jun, Sp-1, AP-1, AP-2, p38, p65Rel, MyD88, IRAK, TRAF6, IκB, inactive NIK, SAP kinase, **SAP-1**, JNK, interferon response genes, NF-κB, Bax, TRAIL, TRAIL receptors, DcR5, TRAIL-R3, TRAIL-R4, RANK, RANK ligand, **Ox40**, **Ox40** ligand, NKG2D, MICA, MICB, NKG2A, NKG2B, NKG2C, NKG2E, NKG2F, TAP1, TAP2 and functional fragments thereof.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> L32 IBIB ABS 1-2
MISSING OPERATOR L32 IBIB
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> D L26 IBIB ABS 1-2

L26 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2007:1175506 CAPLUS
DOCUMENT NUMBER: 147:466839
TITLE: Method for prediction of recurrence of multiple sclerosis
INVENTOR(S): Saito, Toshiro; Sato, Junichi; Yamamura, Takashi
PATENT ASSIGNEE(S): Japan Health Sciences Foundation, Japan
SOURCE: PCT Int. Appl., 32pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2007117020 | A1 | 20071018 | WO 2007-JP57935 | 20070404 |

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: JP 2006-105825 A 20060407

AB Disclosed is a method for prediction of the recurrence of multiple sclerosis. Specifically, the method comprises evaluating the expression level of genes known to vary specifically upon the recurrence of multiple sclerosis, in a peripheral blood CD3+ T lymphocyte in a patient suffering from multiple sclerosis using a DNA microarray (a DNA chip), thereby predicting the recurrence of multiple sclerosis in the patient.

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L26 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Find References

ACCESSION NUMBER: 2001:338762 CAPLUS
DOCUMENT NUMBER: 134:362292
TITLE: Methods of determining individual hypersensitivity to a pharmaceutical agent from gene expression profile
INVENTOR(S): Farr, Spencer
PATENT ASSIGNEE(S): Phase-1 Molecular Toxicology, USA
SOURCE: PCT Int. Appl., 222 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2001032928 | A2 | 20010510 | WO 2000-US30474 | 20001103 |
| WO 2001032928 | A3 | 20020725 | | |

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-165398P P 19991105
US 2000-196571P P 20000411

AB The invention discloses methods, gene databases, gene arrays, protein arrays, and devices that may be used to det. the hypersensitivity of individuals to a given agent, such as drug or other chem., in order to prevent toxic side effects. In one embodiment, methods of identifying

hypersensitivity in a subject by obtaining a gene expression profile of multiple genes assocd. with hypersensitivity of the subject suspected to be hypersensitive, and identifying in the gene expression profile of the subject a pattern of gene expression of the genes assocd. with hypersensitivity are disclosed. The gene expression profile of the subject may be compared with the gene expression profile of a normal individual and a hypersensitive individual. The gene expression profile of the subject that is obtained may comprise a profile of levels of mRNA or cDNA. The gene expression profile may be obtained by using an array of nucleic acid probes for the plurality of genes assocd. with hypersensitivity. The expression of the genes predetd. to be assocd. with hypersensitivity is directly related to prevention or repair of toxic damage at the tissue, organ or system level. Gene databases arrays and app. useful for identifying hypersensitivity in a subject are also disclosed.

OS.CITING REF COUNT: 6 THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (6 CITINGS)

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=>
=> D L32 IBIB ABS 1-3

L32 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | More References

ACCESSION NUMBER: 2009:1626269 CAPLUS
DOCUMENT NUMBER: 152:589804
TITLE: Expressions of activating and inhibitory receptors as well as costimulatory molecules on peripheral blood natural killer cells in patients with recurrent genital herpes
AUTHOR(S): Qian, Qifeng; Zhen, Lin; Li, Qing
CORPORATE SOURCE: Center for STD Control and Research, Shenzhen Institute of Dermatology, Shenzhen, Guangdong Province, 518020, Peop. Rep. China
SOURCE: Zhonghua Pifuke Zazhi (2009), 42(5), 308-310 CODEN: CHFTAJ; ISSN: 0412-4030
PUBLISHER: Zhongguo Yixue Kexueyuan Pifubing Yanjiuso
DOCUMENT TYPE: Journal
LANGUAGE: Chinese

AB The expressions of activating receptors (NKG2D and NKp46), inhibitory receptors (**NKG2A** and KIR) as well as costimulatory mols. (**OX40**, 4-1BB and ICOS) on peripheral blood natural killer (NK) cells from patients with recurrent genital herpes (RGH) were investigated. Four-color immunofluorescence staining with flow cytometry was used to detect the expression of NKG2D, **NKG2A**, KIR and NKp46 in 44 patients with RGH and 40 normal human controls, and to detect the expressions of **OX40**, 4-1BB and ICOS in 29 patients with RGH and 29 normal human controls. The proportions of NKG2D-pos. and NKp46-pos. NK cells significantly decreased in patients with RGH than those in the normal human controls [(93.3±5.4)% vs. (96.9±2.5)%, (88.9±8.7)% vs. (93.4±4.1)%, resp., both P<0.01]. Between the patients and the controls, no significant difference was obsd. in the expression of NK cell inhibitory receptors, **NKG2A** [(41.8±14.4)% vs. (46.0 ± 14.7)%, P>0.05] or KIR [(68.3±19.1)% vs. (69.1±17.6)%, P>0.05]. A lower expression of costimulatory mol. **OX40** was noted in NK cells from patients with RGH compared with those in normal controls [(1.0±1.1)% vs. (1.8±1.7)%, P<0.05]. Herpes simplex virus infection could down-regulate the

expression of NK cell activating receptors and costimulatory mols., subsequently suppress the activation of NK cells, and lead to the escape of virus-infected cells from the killing of NK cells.

L32 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2007:1075452 CAPLUS
DOCUMENT NUMBER: 148:236710
TITLE: Expansion of natural killer cell receptor (CD94/**NKG2A**)-expressing cytolytic CD8 T cells and CD4+CD25+ regulatory T cells from the same cord blood unit
AUTHOR(S): Tanaka, Junji; Sugita, Junichi; Kato, Naoko; Toubai, Tomomi; Ibata, Makoto; Shono, Yusuke; Ota, Shuichi; Kondo, Takeshi; Kobayashi, Takahiko; Kobayashi, Masanobu; Asaka, Masahiro; Imamura, Masahiro
CORPORATE SOURCE: Department of Hematology and Oncology, Institute for Genetic Medicine, Hokkaido University Graduate School of Medicine, Sapporo, Japan
SOURCE: Experimental Hematology (New York, NY, United States) (2007), 35(10), 1562-1566
CODEN: EXHMA6; ISSN: 0301-472X
PUBLISHER: Elsevier Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Objective: Cord blood contains a significant no. of precursor cells that differentiate to cytotoxic effector cells and immunoregulatory cells. We tried to expand inhibitory natural killer cell receptor CD94-expressing CD8 T cells with cytolytic activity and CD4+CD25+ regulatory T cells from the same cord cell unit. Methods: Cytotoxic CD94-expressing CD8 T cells were expanded from CD4-depleted cord blood using an immobilized anti-CD3 monoclonal antibody and a cytokine and also CD4+CD25+ regulatory T cells were expanded from a CD4-enriched fraction derived from the same cord blood unit using anti-CD3/CD28 monoclonal antibody-coated Dynabeads and cytokines. Results: We were able to obtain a more than 1000-fold expansion of CD94-expressing CD8 T cells and a more than 50-fold expansion of CD4+CD25+ cells from the same cord blood unit. These expanded CD4+CD25+ cells expressed FoxP3 mRNA at a level about 100-fold higher than that in isolated CD25- cells and could suppress allogeneic mixed lymphocyte culture by >80% (effector cells: CD4+CD25+ cells = 2:1). Cytolytic activities of purified CD94-expressing cells detected by a 4-h 51Cr release assay against K562 were >60%. Coculture of CD94-expressing cells with expanded CD4+CD25+ cells did not have any effect on cytolytic activities of purified CD94-expressing cells against K562 cells. Conclusion: These expanded cytolytic CD94-expressing CD8 cells might be able to induce a graft-vs-leukemia effect without enhancing graft-vs-host disease, and CD4+CD25+ cells might be able to suppress allogeneic responses, including graft-vs-host disease and graft rejection after cord blood transplantation.

REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L32 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | RE3 References

ACCESSION NUMBER: 2004:1156439 CAPLUS
DOCUMENT NUMBER: 142:73408
TITLE: DNA vaccines comprising immunomodulatory proteins and antigen from pathogens

INVENTOR(S): Weiner, David B.; Muthumani, Karuppiah; Kutzler, Michele; Choo, Andrew K.; Chattergoon, Michael A.
PATENT ASSIGNEE(S): The Trustees of the University of Pennsylvania, USA
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2004112706 | A2 | 20041229 | WO 2004-US19028 | 20040614 |
| WO 2004112706 | A3 | 20050414 | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW | | | | |
| RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| AU 2004249191 | A1 | 20041229 | AU 2004-249191 | 20040614 |
| AU 2004249191 | B2 | 20110106 | | |
| CA 2529051 | A1 | 20041229 | CA 2004-2529051 | 20040614 |
| EP 1633372 | A2 | 20060315 | EP 2004-755303 | 20040614 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK | | | | |
| JP 2007502868 | T | 20070215 | JP 2006-533794 | 20040614 |
| US 20070104686 | A1 | 20070510 | US 2004-560653 | 20040614 |
| PRIORITY APPLN. INFO.: | | | US 2003-478187P P | 20030613 |
| | | | US 2003-478230P P | 20030613 |
| | | | US 2003-478250P P | 20030613 |
| | | | WO 2004-US19028 W | 20040614 |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The authors disclose the use of recombinant vaccines and live attenuated pathogens comprising one or more isolated nucleic acid mols. that encode an immunogen in combination with an isolated nucleic acid mol. that encodes an immunomodulator protein selected from the group consisting of: Fos, c-jun, Sp-1, AP-1, AP-2, p38, p65Rel, MyD88, IRAK, TRAF6, IκB, inactive NIK, SAP kinase, SAP-1, JNK, interferon response genes, NF-κB, Bax, TRAIL, TRAIL receptors, DcR5, TRAIL-R3, TRAIL-R4, RANK, RANK ligand, **Ox40**, **Ox40** ligand, NKG2D, MICA, MICB, **NKG2A**, NKG2B, NKG2C, NKG2E, NKG2F, TAP1, TAP2 and functional fragments thereof.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> D L32 TBIB ABS 4

L32 ANSWER 4 OF 4 BIOSIS COPYRIGHT (c) 2011 The Thomson Corporation on STN

Full Text | More References

ACCESSION NUMBER: 2009:451084 BIOSIS
DOCUMENT NUMBER: PREV200900452187

TITLE: Expression of activating and inhibitory receptors as well as costimulatory molecules on peripheral blood natural killer cells in patients with recurrent genital herpes.
AUTHOR(S): Qian Qi-feng [Reprint Author]; Zhen Lin; Li Qing
CORPORATE SOURCE: Ctr STD Control and Res, Shenzhen Inst Dermatol, Shenzhen 518020, Guangdong, Peoples R China
SOURCE: Zhonghua Pifuke Zazhi, (MAY 2009) Vol. 42, No. 5, pp. 308-310.
CODEN: CHFTAJ. ISSN: 0412-4030.
DOCUMENT TYPE: Article
LANGUAGE: Chinese
ENTRY DATE: Entered STN: 29 Jul 2009
Last Updated on STN: 29 Jul 2009

AB Objective To investigate the expression of activating receptors (NKG2D and NKp46), inhibitory receptors (**NKG2A** and KIR) as well as costimulatory molecules (**OX40**, 4-1BB and ICOS) on peripheral blood natural killer (NK) cells from patients with recurrent genital herpes (RGH). Methods Four-color immunofluorescence staining with flow cytometry was used to detect the expression of NKG2D, **NKG2A**, KIR and NKp46 in 44 patients with RGH and 40 normal human controls, and to detect the expression of **OX40**, 4-1BB and ICOS in 29 patients with RGH and 29 normal human controls. Results The proportions of NKG2D-positive and NKp46-positive NK cells significantly decreased in patients with RGH than those in the normal human controls [(93.3 +/- 5.4)% vs (96.9 +/- 2.5)%, (88.9 +/- 8.7)% vs(93.4 +/- 4.1)%, respectively, both P < 0.011. Between the patients and controls, no significant difference was observed in the expression of NK cell inhibitory receptors, **NKG2A** [(41.8 +/- 14.4)% vs (46.0 +/- 14.7)%, P > 0.05] or KIR [(68.3 +/- 19.1)% vs (69.1 +/- 17.6)%, P > 0.05]. A lower expression of costimulatory molecule **OX40** was noted in NK cells from patients with RGH compared with those in normal controls [(1.0 +/- 1.0% vs 0.8 +/- 1.7)%, P < 0.05]. Conclusions Herpes simplex virus infection could down-regulate the expression of NK cell activating receptors and costimulatory molecules, subsequently suppress the activation of NK cells, and lead to the escape of virus-infected cells from the killing of NK cells.

=> D L24 IBIB ABS 1-3

L24 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2004:1156439 CAPLUS
DOCUMENT NUMBER: 142:73408
TITLE: DNA vaccines comprising immunomodulatory proteins and antigen from pathogens
INVENTOR(S): Weiner, David B.; Muthumani, Karuppiah; Kutzler, Michele; Choo, Andrew K.; Chattergoon, Michael A.
PATENT ASSIGNEE(S): The Trustees of the University of Pennsylvania, USA
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2004112706 | A2 | 20041229 | WO 2004-US19028 | 20040614 |
| WO 2004112706 | A3 | 20050414 | | |

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

| | | | | | |
|---|---|---|---|---|---|
| AU 2004249191 | A1 | 20041229 | AU 2004-249191 | | 20040614 |
| AU 2004249191 | B2 | 20110106 | | | |
| CA 2529051 | A1 | 20041229 | CA 2004-2529051 | | 20040614 |
| EP 1633372 | A2 | 20060315 | EP 2004-755303 | | 20040614 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK | | | | | |
| JP 2007502868 | T | 20070215 | JP 2006-533794 | | 20040614 |
| US 20070104686 | A1 | 20070510 | US 2004-560653 | | 20040614 |
| PRIORITY APPLN. INFO.: | | | US 2003-478187P | P | 20030613 |
| | | | US 2003-478230P | P | 20030613 |
| | | | US 2003-478250P | P | 20030613 |
| | | | WO 2004-US19028 | W | 20040614 |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The authors disclose the use of recombinant vaccines and live attenuated pathogens comprising one or more isolated nucleic acid mols. that encode an immunogen in combination with an isolated nucleic acid mol. that encodes an immunomodulator protein selected from the group consisting of: Fos, c-jun, Sp-1, AP-1, AP-2, p38, p65Rel, MyD88, IRAK, TRAF6, IxB, inactive NIK, SAP kinase, SAP-1, **JNK**, interferon response genes, NF-kB, Bax, TRAIL, TRAIL receptors, DcR5, TRAIL-R3, TRAIL-R4, RANK, RANK ligand, **Ox40**, **Ox40** ligand, NKG2D, MICA, MICB, NKG2A, NKG2B, NKG2C, NKG2E, NKG2F, TAP1, TAP2 and functional fragments thereof.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | *RTG References

ACCESSION NUMBER: 2004:156467 CAPLUS

DOCUMENT NUMBER: 140:198053

TITLE: Expression of CD30 and **Ox40** on T lymphocyte subsets is controlled by distinct regulatory mechanisms

AUTHOR(S): Toennies, Holly M.; Green, Jonathan M.; Arch, Robert H.

CORPORATE SOURCE: Department of Medicine, School of Medicine, Washington University, St. Louis, MO, USA

SOURCE: Journal of Leukocyte Biology (2004), 75(2), 350-357 CODEN: JLBIE7; ISSN: 0741-5400

PUBLISHER: Federation of American Societies for Experimental Biology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Members of the TNF receptor (TNFR) superfamily are cell-surface proteins that can be found on most cell types including lymphocytes. Although some TNFR-related mols. are constitutively expressed, others, such as CD30 and **Ox40**, are induced upon activation of lymphocytes. CD30 and **Ox40** are predominantly expressed on activated T helper (Th)2 cells. Both receptors

can activate c-Jun N-terminal kinase (**JNK**) and nuclear factor-kB (NF-kB) and have been suggested to play costimulatory roles in lymphocyte activation. To gain further insight into events triggered by both TNFR-related mols., a detailed anal. of their expression patterns has been performed. We found that CD30 and **Ox40** were coexpressed on Th2 cells. However, in contrast to CD30, **Ox40** was also expressed on Th1 cells. Although expression of both receptors is augmented by interleukin-4, only CD30 expression is dependent on signal transducer and activator of transcription (STAT)-6-mediated signaling. Differences in the regulatory pathways controlling expression of CD30 and **Ox40** suggest distinct, functional effects triggered by the two TNFR-related mols. during lymphocyte activation.

OS.CITING REF COUNT: 10 THERE ARE 10 CAPLUS RECORDS THAT CITE THIS RECORD (10 CITINGS)

REFERENCE COUNT: 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 3 OF 3 BIOSIS COPYRIGHT (c) 2011 The Thomson Corporation on STN

Full Text | Citing References

ACCESSION NUMBER: 2004:152888 BIOSIS
DOCUMENT NUMBER: PREV200400155749
TITLE: Expression of CD30 and **Ox40** on T lymphocyte subsets is controlled by distinct regulatory mechanisms.
AUTHOR(S): Toennies, Holly M.; Green, Jonathan M.; Arch, Robert H. [Reprint Author]
CORPORATE SOURCE: School of Medicine, Washington University, 660 S. Euclid Ave., Campus Box 8052, Saint Louis, MO, 63110, USA arch@wustl.edu
SOURCE: Journal of Leukocyte Biology, (February 2004) Vol. 75, No. 2, pp. 350-357. print.
ISSN: 0741-5400 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 17 Mar 2004
Last Updated on STN: 17 Mar 2004

AB Members of the TNF receptor (TNFR) superfamily are cell-surface proteins that can be found on most cell types including lymphocytes. Although some TNFR-related molecules are constitutively expressed, others, such as CD30 and **Ox40**, are induced upon activation of lymphocytes. CD30 and **Ox40** are predominantly expressed on activated T helper (Th)2 cells. Both receptors can activate c-Jun N-terminal kinase (**JNK**) and nuclear factor-kappaB (NF-kappaB) and have been suggested to play costimulatory roles in lymphocyte activation. To gain further insight into events triggered by both TNFR-related molecules, a detailed analysis of their expression patterns has been performed. We found that CD30 and **Ox40** were coexpressed on Th2 cells. However, in contrast to CD30, **Ox40** was also expressed on Th1 cells. Although expression of both receptors is augmented by interleukin-4, only CD30 expression is dependent on signal transducer and activator of transcription (STAT)-6-mediated signaling. Differences in the regulatory pathways controlling expression of CD30 and **Ox40** suggest distinct, functional effects triggered by the two TNFR-related molecules during lymphocyte activation.

=> D L28 IBIB ABS 1-6

L28 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2011:51492 CAPLUS
DOCUMENT NUMBER: 154:152967
TITLE: Method for generating aptamers with improved off-rates for histology reagents
INVENTOR(S): Zichi, Dominic; Wilcox, Sheri K.; Bock, Chris; Schneider, Daniel J.; Eaton, Bruce; Gold, Larry; Jarvis, Thale C.; Carter, Jeffrey D.
PATENT ASSIGNEE(S): Somalogic, Inc., USA
SOURCE: PCT Int. Appl., 134pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 8
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2011006075 | A1 | 20110113 | WO 2010-US41540 | 20100709 |
| W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW | | | | |
| RW: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | | |
| US 20100055695 | A1 | 20100304 | US 2009-499967 | 20090709 |
| PRIORITY APPLN. INFO.: | | | US 2009-499967 | A 20090709 |
| | | | US 2007-623535 | B2 20070116 |
| | | | US 2007-623580 | A2 20070116 |
| | | | US 2007-950281P | P 20070717 |
| | | | US 2007-950283P | P 20070717 |
| | | | US 2007-950293P | P 20070717 |
| | | | US 2008-31420P | P 20080226 |
| | | | US 2008-51594P | P 20080508 |
| | | | US 2008-175434 | A2 20080717 |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The present disclosure describes the identification and use of aptamers and photoaptamers having slower dissocn. rate consts. than those obtained using previously described methods. Specifically, the present disclosure describes methods for the identification and use of aptamers to one or more targets within a histol. or cytol. sample, which have slow rates of dissocn. The aptamers may be used to assess localization, relative d., and presence or absence of one or more targets in cytol. and histol. samples. Targets may be selected that are specific and diagnostic of a given disease state for which the sample was collected. The aptamers may also be used to introduce target specific signal moieties. In addn. to target identification, the aptamers may be used to amplify signal generation through a variety of methods. High affinity 5-(N-benzylcarboxyamide)-dUTP-contg. aptamers to Her2 were generated. Aptamer 2616-24 had an equil. binding const. of 1.5x10-8 M. The aptamer was synthesized with 5' addn. of a biotin Cy3 label and used to stain HER2 protein in frozen breast carcinoma tissue sections. In the presence of 1 mM dextran sulfate, the HER2 aptamer bound to cell membranes in the expected morphol. pattern in frozen breast tumors that had been classified by immunohistochem. (IHC) as having 3+ HER2 expression, but it did not

bind to breast tumors classified by IHC as 0/neg., or non-breast neg. control tissues.
REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L28 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN
Full Text | Citing References

ACCESSION NUMBER: 2009:1626269 CAPLUS
DOCUMENT NUMBER: 152:589804
TITLE: Expressions of activating and inhibitory receptors as well as costimulatory molecules on peripheral blood natural killer cells in patients with recurrent genital herpes
AUTHOR(S): Qian, Qifeng; Zhen, Lin; Li, Qing
CORPORATE SOURCE: Center for STD Control and Research, Shenzhen Institute of Dermatology, Shenzhen, Guangdong Province, 518020, Peop. Rep. China
SOURCE: Zhonghua Pifuke Zazhi (2009), 42(5), 308-310
CODEN: CHFTAJ; ISSN: 0412-4030
PUBLISHER: Zhongguo Yixue Kexueyuan Pifubing Yanjiuso
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB The expressions of activating receptors (**NKG2D** and NKp46), inhibitory receptors (NKG2A and KIR) as well as costimulatory mols. (**OX40**, 4-1BB and ICOS) on peripheral blood natural killer (NK) cells from patients with recurrent genital herpes (RGH) were investigated. Four-color immunofluorescence staining with flow cytometry was used to detect the expression of **NKG2D**, NKG2A, KIR and NKp46 in 44 patients with RGH and 40 normal human controls, and to detect the expressions of **OX40**, 4-1BB and ICOS in 29 patients with RGH and 29 normal human controls. The proportions of **NKG2D**-pos. and NKp46-pos. NK cells significantly decreased in patients with RGH than those in the normal human controls [(93.3±5.4)% vs. (96.9±2.5)%, (88.9±8.7)% vs. (93.4±4.1)%, resp., both P<0.01]. Between the patients and the controls, no significant difference was obsd. in the expression of NK cell inhibitory receptors, NKG2A [(41.8±14.4)% vs. (46.0 ± 14.7)%, P>0.05] or KIR [(68.3±19.1)% vs. (69.1±17.6)%, P>0.05]. A lower expression of costimulatory mol. **OX40** was noted in NK cells from patients with RGH compared with those in normal controls [(1.0±1.1)% vs. (1.8±1.7)%, P<0.05]. Herpes simplex virus infection could down-regulate the expression of NK cell activating receptors and costimulatory mols., subsequently suppress the activation of NK cells, and lead to the escape of virus-infected cells from the killing of NK cells.

L28 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN
Full Text | Citing References

ACCESSION NUMBER: 2004:1156439 CAPLUS
DOCUMENT NUMBER: 142:73408
TITLE: DNA vaccines comprising immunomodulatory proteins and antigen from pathogens
INVENTOR(S): Weiner, David B.; Muthumani, Karuppiah; Kutzler, Michele; Choo, Andrew K.; Chattergoon, Michael A.
PATENT ASSIGNEE(S): The Trustees of the University of Pennsylvania, USA
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2004112706 | A2 | 20041229 | WO 2004-US19028 | 20040614 |
| WO 2004112706 | A3 | 20050414 | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW | | | | |
| RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| AU 2004249191 | A1 | 20041229 | AU 2004-249191 | 20040614 |
| AU 2004249191 | B2 | 20110106 | | |
| CA 2529051 | A1 | 20041229 | CA 2004-2529051 | 20040614 |
| EP 1633372 | A2 | 20060315 | EP 2004-755303 | 20040614 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK | | | | |
| JP 2007502868 | T | 20070215 | JP 2006-533794 | 20040614 |
| US 20070104686 | A1 | 20070510 | US 2004-560653 | 20040614 |

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|---|---|---|---|
| PRIORITY APPLN. INFO.: | US 2003-478187P | P | 20030613 |
| | US 2003-478230P | P | 20030613 |
| | US 2003-478250P | P | 20030613 |
| | WO 2004-US19028 | W | 20040614 |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The authors disclose the use of recombinant vaccines and live attenuated pathogens comprising one or more isolated nucleic acid mols. that encode an immunogen in combination with an isolated nucleic acid mol. that encodes an immunomodulator protein selected from the group consisting of: Fos, c-jun, Sp-1, AP-1, AP-2, p38, p65Rel, MyD88, IRAK, TRAF6, IκB, inactive NIK, SAP kinase, SAP-1, JNK, interferon response genes, NF-κB, Bax, TRAIL, TRAIL receptors, DcR5, TRAIL-R3, TRAIL-R4, RANK, RANK ligand, **Ox40**, **Ox40** ligand, **NKG2D**, MICA, MICB, NKG2A, NKG2B, NKG2C, NKG2E, NKG2F, TAP1, TAP2 and functional fragments thereof.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L28 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

| | |
|---|---|
| ACCESSION NUMBER: | 2004:741667 CAPLUS |
| DOCUMENT NUMBER: | 141:259352 |
| TITLE: | Cross-Talk between Activated Human NK Cells and CD4+ T Cells via **OX40**-**OX40** Ligand Interactions |
| AUTHOR(S): | Zingoni, Alessandra; Sornasse, Thierry; Cocks, Benjamin G.; Tanaka, Yuetsu; Santoni, Angela; Lanier, Lewis L. |
| CORPORATE SOURCE: | Department of Microbiology and Immunology and the Cancer Research Institute, University of California, San Francisco, CA, 94143, USA |
| SOURCE: | Journal of Immunology (2004), 173(6), 3716-3724 CODEN: JOIMA3; ISSN: 0022-1767 |
| PUBLISHER: | American Association of Immunologists |

DOCUMENT TYPE: Journal
LANGUAGE: English
AB It is important to understand which mols. are relevant for linking innate and adaptive immune cells. In this study, we show that **OX40** ligand is selectively induced on IL-2, IL-12, or IL-15-activated human NK cells following stimulation through **NKG2D**, the low affinity receptor for IgG (CD16) or killer cell Ig-like receptor 2DS2. CD16-activated NK cells costimulate TCR-induced proliferation, and IFN-γ produced by autologous CD4+ T cells and this process is dependent upon expression of **OX40** ligand and B7 by the activated NK cells. These findings suggest a novel and unexpected link between the natural and specific immune responses, providing direct evidence for cross-talk between human CD4+ T cells and NK receptor-activated NK cells.
OS.CITING REF COUNT: 80 THERE ARE 80 CAPLUS RECORDS THAT CITE THIS RECORD (80 CITINGS)
REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L28 ANSWER 5 OF 6 BIOSIS COPYRIGHT (c) 2011 The Thomson Corporation on STN

Full Text | Citing References

ACCESSION NUMBER: 2009:451084 BIOSIS
DOCUMENT NUMBER: PREV200900452187
TITLE: Expression of activating and inhibitory receptors as well as costimulatory molecules on peripheral blood natural killer cells in patients with recurrent genital herpes.
AUTHOR(S): Qian Qi-feng [Reprint Author]; Zhen Lin; Li Qing
CORPORATE SOURCE: Ctr STD Control and Res, Shenzhen Inst Dermatol, Shenzhen 518020, Guangdong, Peoples R China
SOURCE: Zhonghua Pifuke Zazhi, (MAY 2009) Vol. 42, No. 5, pp. 308-310.
CODEN: CHFTAJ. ISSN: 0412-4030.
DOCUMENT TYPE: Article
LANGUAGE: Chinese
ENTRY DATE: Entered STN: 29 Jul 2009
Last Updated on STN: 29 Jul 2009
AB Objective To investigate the expression of activating receptors (**NKG2D** and NKp46), inhibitory receptors (NKG2A and KIR) as well as costimulatory molecules (**OX40**, 4-1BB and ICOS) on peripheral blood natural killer (NK) cells from patients with recurrent genital herpes (RGH). Methods Four-color immunofluorescence staining with flow cytometry was used to detect the expression of **NKG2D**, NKG2A, KIR and NKp46 in 44 patients with RGH and 40 normal human controls, and to detect the expression of **OX40**, 4-1BB and ICOS in 29 patients with RGH and 29 normal human controls. Results The proportions of **NKG2D**-positive and NKp46-positive NK cells significantly decreased in patients with RGH than those in the normal human controls [(93.3 +/- 5.4)% vs (96.9 +/- 2.5)%, (88.9 +/- 8.7)% vs(93.4 +/- 4.1)%, respectively, both $P < 0.011$. Between the patients and controls, no significant difference was observed in the expression of NK cell inhibitory receptors, NKG2A [(41.8 +/- 14.4)% vs (46.0 +/- 14.7)%, $P > 0.05$] or KIR [(68.3 +/- 19.1)% vs (69.1 +/- 17.6)%, $P > 0.05$]. A lower expression of costimulatory molecule **OX40** was noted in NK cells from patients with RGH compared with those in normal controls [(1.0 +/- 1.0% vs 0.8 +/- 1.7)%, $P < 0.05$]. Conclusions Herpes simplex virus infection could down-regulate the expression of NK cell activating receptors and costimulatory molecules, subsequently suppress the activation of NK cells, and lead to the escape of virus-infected cells from the killing of NK cells.

L28 ANSWER 6 OF 6 BIOSIS COPYRIGHT (c) 2011 The Thomson Corporation on STN

Full Text | String References

ACCESSION NUMBER: 2005:1507 BIOSIS
DOCUMENT NUMBER: PREV200500003849
TITLE: Cross-talk between activated human NK cells and CD4+ T cells via **OX40**-**OX40** ligand interactions.
AUTHOR(S): Zingoni, Alessandra; Sornasse, Thierry; Cocks, Benjamin G.; Tanaka, Yuetsu; Santoni, Angela; Lanier, Lewis L. [Reprint Author]
CORPORATE SOURCE: Dept Microbiol and Immunol, Univ Calif San Francisco, 513 Parnassus Ave, San Francisco, CA, 94143, USA
lanier@itsa.ucsf.edu
SOURCE: Journal of Immunology, (September 15 2004) Vol. 173, No. 6, pp. 3716-3724. print.
ISSN: 0022-1767 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Dec 2004
Last Updated on STN: 16 Dec 2004

AB It is important to understand which molecules are relevant for linking innate and adaptive immune cells. In this study, we show that **OX40** ligand is selectively induced on IL-2, IL-12, or IL-15-activated human NK cells following stimulation through **NKG2D**, the low affinity receptor for IgG (CD16) or killer cell Ig-like receptor 2DS2. CD16-activated NK cells costimulate TCR-induced proliferation, and IFN-gamma produced by autologous CD4+ T cells and this process is dependent upon expression of **OX40** ligand and B7 by the activated NK cells. These findings suggest a novel and unexpected link between the natural and specific immune responses, providing direct evidence for cross-talk between human CD4+ T cells and NK receptor-activated NK cells.

=> D L22 IBIB AES 1-6

L22 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | String References

ACCESSION NUMBER: 2010:1188209 CAPLUS
DOCUMENT NUMBER: 153:429194
TITLE: Aptamer-targeted siRNA to prevent attenuation or suppression of a T cell function
INVENTOR(S): Gilboa, Eli
PATENT ASSIGNEE(S): University of Miami, USA
SOURCE: U.S. Pat. Appl. Publ., 46pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| US 20100240732 | A1 | 20100923 | US 2010-752802 | 20100401 |
| PRIORITY APPLN. INFO.: | | | WO 2008-US78455 A | 20081001 |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Compns. for countering immune attenuating/suppressive pathways comprise targeting agents or aptamer-targeted RNAi-mediated gene silencing (siRNA/shRNA). The targeting agents or aptamers are specific for immune cells and markers thereof, including mols. comprising 4-1BB (CD137), **OX40**, CD3, CD28, HLA-ABC, HLA-DR, T cell receptor αβ, T cell

receptor γδ, T cell receptor ζ, TGFβRII, TNF receptors, CD11c, CD1-339, B7, mannose receptor, or DEC205. The RNAi is specific for any one or more polynucleotides comprising TGFβ receptor, TGFβRII, polynucleotides assocd. with TGFβ signaling, purinergic receptors, CTLA-4, PTEN, Csk, Cbl-b, cytokines, SOCS1, GILT, GILZ, A20, or **Bax**/Bak. These aptamer-RNAi fusion compns. (e.g., a 4-1BB dimer aptamer-CTLA-4 siRNA fusion) have broad applicability in the treatment of many diseases.

L22 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2004:1156439 CAPLUS
DOCUMENT NUMBER: 142:73408
TITLE: DNA vaccines comprising immunomodulatory proteins and antigen from pathogens
INVENTOR(S): Weiner, David B.; Muthumani, Karuppiah; Kutzler, Michele; Choo, Andrew K.; Chattergoon, Michael A.
PATENT ASSIGNEE(S): The Trustees of the University of Pennsylvania, USA
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2004112706 | A2 | 20041229 | WO 2004-US19028 | 20040614 |
| WO 2004112706 | A3 | 20050414 | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW | | | | |
| RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| AU 2004249191 | A1 | 20041229 | AU 2004-249191 | 20040614 |
| AU 2004249191 | B2 | 20110106 | | |
| CA 2529051 | A1 | 20041229 | CA 2004-2529051 | 20040614 |
| EP 1633372 | A2 | 20060315 | EP 2004-755303 | 20040614 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK | | | | |
| JP 2007502868 | T | 20070215 | JP 2006-533794 | 20040614 |
| US 20070104686 | A1 | 20070510 | US 2004-560653 | 20040614 |
| PRIORITY APPLN. INFO.: | | | US 2003-478187P P | 20030613 |
| | | | US 2003-478230P P | 20030613 |
| | | | US 2003-478250P P | 20030613 |
| | | | WO 2004-US19028 W | 20040614 |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The authors disclose the use of recombinant vaccines and live attenuated pathogens comprising one or more isolated nucleic acid mols. that encode an immunogen in combination with an isolated nucleic acid mol. that encodes an immunomodulator protein selected from the group consisting of: Fos, c-jun, Sp-1, AP-1, AP-2, p38, p65Rel, MyD88, IRAK, TRAF6, IκB, inactive NIK, SAP kinase, SAP-1, JNK, interferon response genes,

NF-κB, **Bax**, TRAIL, TRAIL receptors, DcR5, TRAIL-R3, TRAIL-R4, RANK, RANK ligand, **Ox40**, **Ox40** ligand, NKG2D, MICA, MICB, NKG2A, NKG2B, NKG2C, NKG2E, NKG2F, TAP1, TAP2 and functional fragments thereof.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2004:672497 CAPLUS
DOCUMENT NUMBER: 141:241995
TITLE: Functional expression of CD134 by neutrophils
AUTHOR(S): Baumann, Ralf; Yousefi, Shida; Simon, Dagmar; Russmann, Stefan; Mueller, Christoph; Simon, Hans-Uwe
CORPORATE SOURCE: Department of Pharmacology, University of Bern, Bern, Switz.
SOURCE: European Journal of Immunology (2004), 34(8), 2268-2275
CODEN: EJIMAF; ISSN: 0014-2980
PUBLISHER: Wiley-VCH Verlag GmbH & Co. KGaA
DOCUMENT TYPE: Journal
LANGUAGE: English

AB CD134 (**OX40**) is a member of the tumor necrosis factor (TNF) receptor superfamily expressed on activated T cells. Here, the authors show that human peripheral blood neutrophils express CD134. Activation of CD134 by sol. CD134 ligand (**OX40** ligand/gp34) resulted in delayed caspase-3 activation and consequently in delayed neutrophil apoptosis in vitro. Moreover, CD134 ligand, like G-CSF, maintained anti-apoptotic Mcl-1 levels and inhibited cleavage of the pro-apoptotic Bcl-2 family members Bid and **Bax** in these cells, suggesting that CD134-mediated signals block apoptosis pathways proximal to mitochondria activation. In conclusion, CD134 regulates neutrophil survival, suggesting that this mol. does not only contribute to adaptive but also to innate immune responses.

OS.CITING REF COUNT: 18 THERE ARE 18 CAPLUS RECORDS THAT CITE THIS RECORD (18 CITINGS)

REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2003:324436 CAPLUS
DOCUMENT NUMBER: 139:147741
TITLE: Oncogene expression on the syngeneic β-cells of long-term surviving pancreatic grafts and better effects of interleukin-1 receptor (IL-1R) and IL-2Rβ on the grafted β-cells in LEW/Sea strain rats
AUTHOR(S): Nakatsuji, Tadako
CORPORATE SOURCE: Department of Transfusion, Hamamatsu University School of Medicine, Hamamatsu, 431-3192, Japan
SOURCE: Transplant Immunology (2003), 11(1), 49-56
CODEN: TRIME2; ISSN: 0966-3274
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Thirty-two normal LEW/Sea rats were transplanted a piece of syngeneic pancreas between the peritoneum and abdominal muscle. Among them, 17

(68%) of the 25 rats that received pancreatic transplantation at 41-50 days of age had a surviving β-cell mass at 5.5-7.1 mo after transplantation. Among the 25 rats, 12 rats injected with interleukin-1 receptor (IL-1R) and IL-2Rβ peptides at post-transplantation showed better surviving grafts at 5.5 mo observation. Only 2 (25%) of the other 7 young rats that received a pancreatic graft at 20 days of age had a small mass at 21 days post-transplantation. Flow cytometer (FCM) analyses showed that thymus **OX40**+ (CD134+) T-cells were increased up to 37% at the graft rejection in the 13 old rats without the IL-R peptide injections. The 7 young rats had 99% of thymus **OX40**+ T-cells. However, the 12 old rats injected with the IL-R peptides showed suppressed nos. of thymus **OX40**+ T-cells (8-13%). The long-term surviving, but apoptotic, grafted β-cells were stained pos. both with anti-insulin monoclonal antibody (mAb) and with anti-c-erbB-2/human epidermal growth factor receptor (HER)-2/neu mAb. Expression of a c-erb family oncogene was shown on the pancreatic graft surviving for 7.1 mo. Electron microscopic anal. of the grafted β-cells showed abnormally large β granules and loss of functioning mitochondria in the cytoplasm. In 18 (56%) of the 32 rats, the 220-bp and 380-bp specific products of insulin-degrading enzyme (IDE) gene were amplified using the polymerase chain reaction (PCR) of the liver DNA. Among the 18 rats, 6 rats expressed 2 extra bands of 280-bp and 700-bp in a correlation with the high levels of the transforming growth factor-alpha (TGF-α) cDNA of 120-bp which was amplified in the quant. reverse-transcriptase (RT)-PCR of the liver cDNA. Among the selected 11 rats, 5 rats showed large amts. of the 120-bp TGF-α cDNA. Host pancreatic RT-PCR showed 235-bp or 250-bp bcl-2 and 181-bp bcl-xS gene products. The bcl-2 cDNA of the host pancreas was amplified actively when the pancreatic graft was being rejected. Exceptionally, the one female injected with the IL-R peptides showed a low level of the liver TGF-α cDNA together with the pancreatic expressions of **Bax** (140-bp), bcl-2 and like interleukin converting enzyme (LICE) (318-bp) cDNA. Insulin secretion from the grafted β-cells and IL-1β-induced Fas-mediated apoptosis of the β-cells were suspected to be present at the same time in the female with the best graft survival.

REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | *Rfrnc References

ACCESSION NUMBER: 2001:338762 CAPLUS
DOCUMENT NUMBER: 134:362292
TITLE: Methods of determining individual hypersensitivity to a pharmaceutical agent from gene expression profile
INVENTOR(S): Farr, Spencer
PATENT ASSIGNEE(S): Phase-1 Molecular Toxicology, USA
SOURCE: PCT Int. Appl., 222 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2001032928 | A2 | 20010510 | WO 2000-US30474 | 20001103 |
| WO 2001032928 | A3 | 20020725 | | |

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,

HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-165398P P 19991105
US 2000-196571P P 20000411

AB The invention discloses methods, gene databases, gene arrays, protein arrays, and devices that may be used to det. the hypersensitivity of individuals to a given agent, such as drug or other chem., in order to prevent toxic side effects. In one embodiment, methods of identifying hypersensitivity in a subject by obtaining a gene expression profile of multiple genes assocd. with hypersensitivity of the subject suspected to be hypersensitive, and identifying in the gene expression profile of the subject a pattern of gene expression of the genes assocd. with hypersensitivity are disclosed. The gene expression profile of the subject may be compared with the gene expression profile of a normal individual and a hypersensitive individual. The gene expression profile of the subject that is obtained may comprise a profile of levels of mRNA or cDNA. The gene expression profile may be obtained by using an array of nucleic acid probes for the plurality of genes assocd. with hypersensitivity. The expression of the genes predetd. to be assocd. with hypersensitivity is directly related to prevention or repair of toxic damage at the tissue, organ or system level. Gene databases arrays and app. useful for identifying hypersensitivity in a subject are also disclosed.

OS.CITING REF COUNT: 6 THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (6 CITINGS)

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 6 OF 6 BIOSIS COPYRIGHT (c) 2011 The Thomson Corporation on STN

Full Text | Citing References

ACCESSION NUMBER: 2005:456129 BIOSIS
DOCUMENT NUMBER: PREV200510249472
TITLE: Functional expression of CD134 by neutrophils.
AUTHOR(S): Baumann, Ralf; Yousefi, Shida; Simon, Dagmar; Russmann, Stefan; Mueller, Christoph; Simon, Hans-Uwe [Reprint Author]
CORPORATE SOURCE: Univ Bern, Dept Pharmacol, Friedbuhlstr 49, CH-3010 Bern, Switzerland
hus@pki.unibe.ch
SOURCE: European Journal of Immunology, (AUG 2004) Vol. 34, No. 8, pp. 2268-2275.
CODEN: EJIMAF. ISSN: 0014-2980.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Nov 2005
Last Updated on STN: 9 Nov 2005

AB CD134 (**OX40**) is a member of the tumor necrosis factor (TNF) receptor superfamily expressed on activated T cells. Here, we show that human peripheral blood neutrophils express CD134. Activation of CD134 by soluble CD134 ligand (**OX40** ligand/gp34) resulted in delayed caspase-3 activation and consequently in delayed neutrophil apoptosis in vitro. Moreover, CD134 ligand,like G-CSF, maintained anti-apoptotic Mcl-1 levels and inhibited cleavage of the pro-apoptotic Bcl-2 family members Bid and **Bax** in these cells, suggesting that CD134-mediated signals block

apoptosis pathways proximal to mitochondria activation. In conclusion, CD134 regulates neutrophil survival, suggesting that this molecule does not only contribute to adaptive but also to innate immune responses.

=> D L19 IBIB ABS 1-19

L19 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Other References

ACCESSION NUMBER: 2006:987282 CAPLUS
DOCUMENT NUMBER: 145:503953
TITLE: Bbt-TNFR1 and Bbt-TNFR2, two tumor necrosis factor receptors from Chinese amphioxus involve in host defense
AUTHOR(S): Yuan, Shaochun; Yu, Yanhong; Huang, Shengfeng; Liu, Tong; Wu, Tao; Dong, Meiling; Chen, Shangwu; Yu, Yingcai; Xu, Anlong
CORPORATE SOURCE: State Key Laboratory of Biocontrol, Department of Biochemistry, Open Laboratory for Marine Functional Genomics of State High-Tech Development Program, Guangdong Key Laboratory of Therapeutic Functional Genes, College of Life Sciences, Sun Yat-Sen (Zhongshan) University, Guangzhou, 510275, Peop. Rep. China
SOURCE: Molecular Immunology (2007), 44(5), 756-762 CODEN: MOIMD5; ISSN: 0161-5890
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Two novel tumor necrosis factor receptors, Bbt-TNFR1 and Bbt-TNFR2, were isolated from Chinese amphioxus, the closest relative to vertebrate. The mRNA of Bbt-TNFR1 encoded a type I membrane protein of 452 amino acids, including 4 cysteine-rich domains in the extracellular region and a putative **TRAF6**-binding site at its 154 amino acid (aa) long cytoplasmic tail. Bbt-TNFR2 was a 304 aa long type I membrane protein, featuring 3 cysteine-rich domains and a short cytoplasmic tail of just 13 aa. Southern blot revealed that Bbt-TNFR1 was a single copy gene, while Bbt-TNFR2 was presented in multiple copies. Sequence comparison indicated that both Bbt-TNFR1 and Bbt-TNFR2 were weakly similar to LT-bR, HVEM, TNFR2, CD40, **OX40**, and DcR3. Real-time PCR showed that Bbt-TNFR1 and Bbt-TNFR2 were regulated during development and finally had high expression in mucosa-rich tissues in adult stage. Furthermore, up-regulated expression of both genes was also obsd. in gut after Gram-pos. bacteria challenge. However, not like Bbt-TNFR2 slow and gradual augmentation in the following 48 h, expression of Bbt-TNFR1 dramatically surged up within 4 h and then subsided rapidly. Thus, Bbt-TNFR1 and Bbt-TNFR2 may be involved in the host defense of Chinese amphioxus via distinct fashions.

OS.CITING REF COUNT: 6 THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (6 CITINGS)
REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L19 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Other References

ACCESSION NUMBER: 2005:200736 CAPLUS
DOCUMENT NUMBER: 142:278479
TITLE: TNF receptor (TNFR)-associated factor (TRAF) 3 serves

as an inhibitor of TRAF2/5-mediated activation of the noncanonical NF-κB pathway by TRAF-binding TNFRs

AUTHOR(S): Hauer, Julia; Pueschner, Stephanie; Ramakrishnan, Parameswaran; Simon, Ute; Bongers, Martina; Federle, Christine; Engelmann, Hartmut

CORPORATE SOURCE: Institut fuer Immunologie der Universitaet Muenchen, Munich, 80366, Germany

SOURCE: Proceedings of the National Academy of Sciences of the United States of America (2005), 102(8), 2874-2879 CODEN: PNASA6; ISSN: 0027-8424

PUBLISHER: National Academy of Sciences

DOCUMENT TYPE: Journal

LANGUAGE: English

AB TNF family members and their receptors contribute to increased gene expression for inflammatory processes and intracellular cascades leading to programmed cell death, both via activation of NF-κB. TNF receptor (TNFR)-assocd. factors (TRAFs) are cytoplasmic adaptor proteins binding to various receptors of the TNFR family. In an attempt to delineate the role of individual TRAFs, we compared NF-κB activation by CD40wt and CD40 mutants with different TRAF recruitment patterns. Recognized only recently, NF-κB signaling occurs at least via two different pathways. Each pathway results in nuclear translocation of two different Rel-dimers, the canonical p50/RelA and the noncanonical p52/RelB. Here, we show that via **TRAF6**, CD40 mediates only the activation of the canonical NF-κB pathway. Via TRAF2/5, CD40 activates both the canonical and the noncanonical NF-κB pathways. We obsd. that TRAF3 specifically blocked the NF-κB activation via TRAF2/5. This inhibitory effect of TRAF3 depends on the presence of an intact zinc finger domain. Paradoxically, suppression of TRAF2/5-mediated NF-κB activation by TRAF3 resulted in enhanced transcriptional activity of **TRAF6**-mediated canonical NF-κB emanating from CD40. We also obsd. that 12 TNFR family members (p75TNFR, LTβR, RANK, HVEM, CD40, CD30, CD27, 4-1BB, GITR, BCMA, **OX40**, and TACI) are each capable of activating the alternative NF-κB pathway and conclude that TRAF3 serves as a neg. regulator of this pathway for all tested receptors.

OS.CITING REF COUNT: 75 THERE ARE 75 CAPLUS RECORDS THAT CITE THIS RECORD (75 CITINGS)

REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L19 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | eFirst | References

ACCESSION NUMBER: 2004:1156439 CAPLUS

DOCUMENT NUMBER: 142:73408

TITLE: DNA vaccines comprising immunomodulatory proteins and antigen from pathogens

INVENTOR(S): Weiner, David B.; Muthumani, Karuppiah; Kutzler, Michele; Choo, Andrew K.; Chattergoon, Michael A.

PATENT ASSIGNEE(S): The Trustees of the University of Pennsylvania, USA

SOURCE: PCT Int. Appl., 47 pp. CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2004112706 | A2 | 20041229 | WO 2004-US19028 | 20040614 |

WO 2004112706 A3 20050414
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

| | | | | | |
|---|---|---|---|---|---|
| AU 2004249191 | A1 | 20041229 | AU 2004-249191 | | 20040614 |
| AU 2004249191 | B2 | 20110106 | | | |
| CA 2529051 | A1 | 20041229 | CA 2004-2529051 | | 20040614 |
| EP 1633372 | A2 | 20060315 | EP 2004-755303 | | 20040614 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK | | | | | |
| JP 2007502868 | T | 20070215 | JP 2006-533794 | | 20040614 |
| US 20070104686 | A1 | 20070510 | US 2004-560653 | | 20040614 |
| PRIORITY APPLN. INFO.: | | | US 2003-478187P | P | 20030613 |
| | | | US 2003-478230P | P | 20030613 |
| | | | US 2003-478250P | P | 20030613 |
| | | | WO 2004-US19028 | W | 20040614 |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The authors disclose the use of recombinant vaccines and live attenuated pathogens comprising one or more isolated nucleic acid mols. that encode an immunogen in combination with an isolated nucleic acid mol. that encodes an immunomodulator protein selected from the group consisting of: Fos, c-jun, Sp-1, AP-1, AP-2, p38, p65Rel, MyD88, IRAK, **TRAF6**, IkB, inactive NIK, SAP kinase, SAP-1, JNK, interferon response genes, NF-κB, Bax, TRAIL, TRAIL receptors, DcR5, TRAIL-R3, TRAIL-R4, RANK, RANK ligand, **Ox40**, **Ox40** ligand, NKG2D, MICA, MICB, NKG2A, NKG2B, NKG2C, NKG2E, NKG2F, TAP1, TAP2 and functional fragments thereof.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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Full Text | Citing References

ACCESSION NUMBER: 2006:685311 BIOSIS
DOCUMENT NUMBER: PREV200600679580
TITLE: Bbt-TNFR1 and Bbt-TNFR2, two tumor necrosis factor receptors from Chinese amphioxus involve in host defense.
AUTHOR(S): Yuan, Shaochun; Yu, Yanhong; Huang, Shengfeng; Liu, Tong; Wu, Tao; Dong, Melling; Chen, Shangwu; Yu, Yingcai; Xu, Anlong [Reprint Author]
CORPORATE SOURCE: Zhongshan Univ, State Key Lab Biocontrol,Coll Life Sci, Dept Biochem,Guangdong Key Lab Therapeut Funct Ge, Open Lab Marine Funct Genom,State High Tech Dev P, Guangzhou 510275, Peoples R China
ls36@zsu.edu.cn
SOURCE: Molecular Immunology, (FEB 2007) Vol. 44, No. 5, pp. 756-762.
CODEN: MOIMD5. ISSN: 0161-5890.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 6 Dec 2006

Last Updated on STN: 3 Mar 2010

AB Two novel tumor necrosis factor receptors, Bbt-TNFR1 and Bbt-TNFR2, were isolated from Chinese amphioxus, the closest relative to vertebrate. The mRNA of Bbt-TNFR1 encoded a type I membrane protein of 452 amino acids, including four cysteine-rich domains in the extracellular region and a putative **TRAF6**-binding site at its 154aa long cytoplasmic tail. Bbt-TNFR2 was a 304aa long type I membrane protein, featuring three cysteine-rich domains and a short cytoplasmic tail of just 13 amino acids. Southern blot revealed that Bbt-TNFR1 was a single copy gene, while Bbt-TNFR2 was presented in multiple copies. Sequence comparison indicated that both Bbt-TNFR1 and Bbt-TNFR2 were weakly similar to LT-bR, HVEM, TNFR2, CD40, **OX40** and DcR3. Real-time PCR showed that Bbt-TNFR1 and Bbt-TNFR2 were regulated during development and finally had high expression in mucosa-rich tissues in adult stage. Furthermore, up-regulated expression of both genes was also observed in guts after Gram-positive bacteria challenge. However, not like Bbt-TNFR2's slowly and gradually augmentation in the following 48 h, expression of Bbt-TNFR1 dramatically surged up within 4 It and then subsided rapidly. Taking together, Bbt-TNFR1 and Bbt-TNFR2 may involve in the host defense of Chinese amphioxus via distinct fashions. (c) 2006 Elsevier Ltd. All rights reserved.

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Full Text | String References

ACCESSION NUMBER: 2005:195743 BIOSIS
DOCUMENT NUMBER: PREV200500195658
TITLE: TNF receptor (TNFR)-associated factor (TRAF) 3 serves as an inhibitor of TRAF2/5-mediated activation of the noncanonical NF-kappaB pathway by TRAF-binding TNFRs.
AUTHOR(S): Hauer, Julia; Pueschner, Stephanie; Ramakrishnan, Parameswaran; Simon, Ute; Bongers, Martina; Federle, Christine; Engelmann, Hartmut [Reprint Author]
CORPORATE SOURCE: Inst Immunol, Univ Munich, Goethestr 31, D-80366, Munich, Germany
hengelmann@lmu.de
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (February 22 2005) Vol. 102, No. 8, pp. 2874-2879. print.
ISSN: 0027-8424 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 25 May 2005
Last Updated on STN: 25 May 2005

AB TNF family members and their receptors contribute to increased gene expression for inflammatory processes and intracellular cascades leading to programmed cell death, both via activation of NF-kappaB. TNF receptor (TNFR)-associated factors (TRAFs) are cytoplasmic adaptor proteins binding to various receptors of the TNFR family. In an attempt to delineate the role of individual TRAFs, we compared NF-kappaB activation by CD40wt and CD40 mutants with different TRAF recruitment patterns. Recognized only recently, NF-kappaB signaling occurs at least via two different pathways. Each pathway results in nuclear translocation of two different Rel-dimers, the canonical p50/RelA and the noncanonical p52/ReIB. Here, we show that via TRAM, CD40 mediates only the activation of the canonical NF-kappaB pathway. Via TRAF2/5, CD40 activates both the canonical and the noncanonical NF-kappaB pathways. We observed that TRAF3 specifically blocked the NF-kappaB activation via TRAF2/5. This inhibitory effect of TRAF3 depends on the presence of an intact zinc finger domain. Paradoxically, suppression of TRAF2/5-mediated NF-kappaB activation by

TRAF3 resulted in enhanced transcriptional activity of **TRAF6**-mediated canonical NF-kappaB emanating from CD40. We also observed that 12 TNFR family members (p75TNFR, LTbetaR, RANK, HVEM, CD40, CD30, CD27,4-1BB, GITR, BCMA, **OX40**, and TACI) are each capable of activating the alternative NF-kappaB pathway and conclude that TRAF3 serves as a negative regulator of this pathway for all tested receptors.

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Full Text | Citing References

ACCESSION NUMBER: 2001:332906 BIOSIS
DOCUMENT NUMBER: PREV200100332906
TITLE: Chronic lymphocytic leukemia B cells impair immunoglobulin class switching by dysregulating a CD30+ T cell-dependent CD40-inhibitory pathway.
AUTHOR(S): Cerutti, Andrea [Reprint author]; Schaffer, Andras [Reprint author]; Casali, Paolo [Reprint author]
CORPORATE SOURCE: Department of Pathology, Division of Molecular Immunology, Weill Medical College of Cornell University, New York, NY, USA
SOURCE: Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 472a. print.
Meeting Info.: 42nd Annual Meeting of the American Society of Hematology. San Francisco, California, USA. December 01-05, 2000. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Jul 2001
Last Updated on STN: 19 Feb 2002

AB Chronic lymphocytic leukemia (CLL) is a B cell lymphoproliferative disorder associated with impaired Ig class switching from IgM to IgG and IgA, a defect that leads to recurrent bacterial infections. The pathogenesis of this immunodeficiency is poorly understood. Naive B cells undergo class switching upon engagement of CD40 by CD154 (CD40 ligand), a molecule expressed by T cells few hours after activation by antigen. A few days later, T cells express CD30, a physiological negative modulator of the immune response. We show here that, in CLL patients, CD8+ CD28- suppressor T cells are increased and constitutively express CD30. In addition, leukemic CLL B cells rapidly up-regulate CD30 on CD4+ T cells through a CD134L (**OX40** ligand) and IL-4-dependent mechanism. These leukemia-induced CD30+ T cells inhibit class switch DNA recombination (CSR) by engaging CD153 (CD30 ligand) on normal naive B cells. Signals emanating from B cell CD153 interfere with the CD154-induced recruitment of TNF receptor-associated-protein (TRAF)2, TRAF2, TRAF3, TRAF5, **TRAF6** and TNF-associated activator of NF-kappaB (TANK) to CD40. They also inhibit the CD154-induced activation of IkappaB kinase (IKK), the degradation of IkappaB, and the subsequent nuclear translocation of NF-kappaB, a transcription factor critical for CSR to occur. By showing that engagement of T cell CD30 by CD153 on leukemic B cells down-regulates CD154, our findings suggest that, in CLL, dysregulated CD30:CD153 interaction impairs class switching and antibody production by transmitting bidirectional CD40 and CD154-inhibitory signals.

=> D L35 IBIB ABS 1-6

L35 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2004:1156439 CAPLUS
DOCUMENT NUMBER: 142:73408
TITLE: DNA vaccines comprising immunomodulatory proteins and antigen from pathogens
INVENTOR(S): Weiner, David B.; Muthumani, Karuppiah; Kutzler, Michele; Choo, Andrew K.; Chattergoon, Michael A.
PATENT ASSIGNEE(S): The Trustees of the University of Pennsylvania, USA
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2004112706 | A2 | 20041229 | WO 2004-US19028 | 20040614 |
| WO 2004112706 | A3 | 20050414 | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW | | | | |
| RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| AU 2004249191 | A1 | 20041229 | AU 2004-249191 | 20040614 |
| AU 2004249191 | B2 | 20110106 | | |
| CA 2529051 | A1 | 20041229 | CA 2004-2529051 | 20040614 |
| EP 1633372 | A2 | 20060315 | EP 2004-755303 | 20040614 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK | | | | |
| JP 2007502868 | T | 20070215 | JP 2006-533794 | 20040614 |
| US 20070104686 | A1 | 20070510 | US 2004-560653 | 20040614 |
| PRIORITY APPLN. INFO.: | | | US 2003-478187P | P 20030613 |
| | | | US 2003-478230P | P 20030613 |
| | | | US 2003-478250P | P 20030613 |
| | | | WO 2004-US19028 | W 20040614 |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The authors disclose the use of recombinant vaccines and live attenuated pathogens comprising one or more isolated nucleic acid mols. that encode an immunogen in combination with an isolated nucleic acid mol. that encodes an immunomodulator protein selected from the group consisting of: **Fos**, c-jun, Sp-1, AP-1, AP-2, p38, p65Rel, MyD88, IRAK, TRAF6, IκB, inactive NIK, SAP kinase, SAP-1, JNK, interferon response genes, NF-κB, Bax, TRAIL, TRAIL receptors, DcR5, TRAIL-R3, TRAIL-R4, RANK, RANK ligand, **Ox40**, **Ox40** ligand, NKG2D, MICA, MICB, NKG2A, NKG2B, NKG2C, NKG2E, NKG2F, TAP1, TAP2 and functional fragments thereof.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L35 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2003:202915 CAPLUS
DOCUMENT NUMBER: 138:215303
TITLE: Methods for predicting drug sensitivity in patients afflicted with an inflammatory disease
INVENTOR(S): Hakonarson, Hakon
PATENT ASSIGNEE(S): Decode Genetics Ehf., Iceland
SOURCE: PCT Int. Appl., 61 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2003021261 | A2 | 20030313 | WO 2002-IB3613 | 20020902 |
| WO 2003021261 | A3 | 20031120 | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW | | | | |
| RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| US 20030113831 | A1 | 20030619 | US 2001-947991 | 20010906 |
| US 7384736 | B2 | 20080610 | | |
| CA 2457476 | A1 | 20030313 | CA 2002-2457476 | 20020902 |
| AU 2002328110 | A1 | 20030318 | AU 2002-328110 | 20020902 |
| AU 2002328110 | B2 | 20070125 | | |
| EP 1428023 | A2 | 20040616 | EP 2002-762680 | 20020902 |
| EP 1428023 | B1 | 20080827 | | |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK | | | | |
| JP 2005502345 | T | 20050127 | JP 2003-525293 | 20020902 |
| AT 406575 | T | 20080915 | AT 2002-762680 | 20020902 |
| PRIORITY APPLN. INFO.: | | | US 2001-947991 A2 | 20010906 |
| | | | WO 2002-IB3613 W | 20020902 |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Methods are disclosed for predicting the efficacy of a drug for treating an inflammatory disease in a human patient, including: obtaining a sample of cells from the patient; obtaining a gene expression profile of the sample in the absence and presence of in vitro modulation of the cells with specific cytokines and/or mediators; and comparing the gene expression profile of the sample with a ref. gene expression profile, wherein similarities between the sample expression profile and the ref. expression profile predicts the efficacy of the drug for treating the inflammatory disease in the patient.

OS.CITING REF COUNT: 7 THERE ARE 7 CAPLUS RECORDS THAT CITE THIS RECORD (12 CITINGS)

L35 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2002:583793 CAPLUS
DOCUMENT NUMBER: 137:351109

TITLE: Signaling of gp34 (**OX40** ligand) induces vascular endothelial cells to produce a CC chemokine RANTES/CCL5
AUTHOR(S): Kotani, Ai; Hori, Toshiyuki; Matsumura, Yumi; Uchiyama, Takashi
CORPORATE SOURCE: Graduate School of Medicine, Department of Hematology and Oncology, Kyoto University, Kyoto, 606-8507, Japan
SOURCE: Immunology Letters (2002), 84(1), 1-7
CODEN: IMLED6; ISSN: 0165-2478
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The authors previously showed that gp34 (**OX40** ligand) expressed on vascular endothelial cells is not only involved in adhesion between activated T cells and endothelial cells but also by itself able to transmit intracellular signals leading to expression of c-**fos** and c-jun mRNA upon **OX40** binding. In the present study, the authors searched for genes that were induced or upregulated by gp34 signaling in human umbilical vein endothelial cells (HUVECs) to define its downstream biol. events. HUVECs expressing high levels of gp34 were stimulated with recombinant sol. **OX40** or mock control and subjected to anal. using cDNA expression arrays. The authors found that a CC chemokine RANTES (regulated upon activation, normal T cell expressed and secreted)/CCL5 is one of such inducible genes. Reverse transcriptase-PCR anal. showed that expression of RANTES mRNA was induced after incubation with sol. **OX40** and this induction was inhibited by anti-gp34 mAb. The authors could detect expression of intracellular RANTES protein by flow cytometry in HUVECs stimulated with sol. **OX40** as well as fixed **OX40** transfectant cells but not those stimulated with mock supernatants or mock transfectant cells. Again, this induction of RANTES protein was inhibited by anti-gp34 mAb. These results clearly indicate that gp34 signaling induces expression of RANTES at both mRNA and protein levels in HUVECs and suggest a possible link between the **OX40**/gp34 system and RANTES during the process of T cell adhesion to endothelial cells and subsequent extravasation.

OS.CITING REF COUNT: 27 THERE ARE 27 CAPLUS RECORDS THAT CITE THIS RECORD (27 CITINGS)
REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L35 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2001:338762 CAPLUS
DOCUMENT NUMBER: 134:362292
TITLE: Methods of determining individual hypersensitivity to a pharmaceutical agent from gene expression profile
INVENTOR(S): Farr, Spencer
PATENT ASSIGNEE(S): Phase-1 Molecular Toxicology, USA
SOURCE: PCT Int. Appl., 222 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2001032928 | A2 | 20010510 | WO 2000-US30474 | 20001103 |
| WO 2001032928 | A3 | 20020725 | | |

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-165398P P 19991105
US 2000-196571P P 20000411

AB The invention discloses methods, gene databases, gene arrays, protein arrays, and devices that may be used to det. the hypersensitivity of individuals to a given agent, such as drug or other chem., in order to prevent toxic side effects. In one embodiment, methods of identifying hypersensitivity in a subject by obtaining a gene expression profile of multiple genes assocd. with hypersensitivity of the subject suspected to be hypersensitive, and identifying in the gene expression profile of the subject a pattern of gene expression of the genes assocd. with hypersensitivity are disclosed. The gene expression profile of the subject may be compared with the gene expression profile of a normal individual and a hypersensitive individual. The gene expression profile of the subject that is obtained may comprise a profile of levels of mRNA or cDNA. The gene expression profile may be obtained by using an array of nucleic acid probes for the plurality of genes assocd. with hypersensitivity. The expression of the genes predetd. to be assocd. with hypersensitivity is directly related to prevention or repair of toxic damage at the tissue, organ or system level. Gene databases arrays and app. useful for identifying hypersensitivity in a subject are also disclosed.

OS.CITING REF COUNT: 6 THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (6 CITINGS)

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L35 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 1999:589597 CAPLUS
DOCUMENT NUMBER: 131:309660
TITLE: Intracellular signaling of gp34, the **OX40** ligand: induction of c-jun and c-**fos** mRNA expression through gp34 upon binding of its receptor, **OX40**
AUTHOR(S): Matsumura, Yumi; Hori, Toshiyuki; Kawamata, Shin; Imura, Akihiro; Uchiyama, Takashi
CORPORATE SOURCE: Departments of Hematology and Oncology and Dermatology, Graduate School of Medicine, and Research Center for Acquired Immunodeficiency Syndrome, The Institute for Virus Research, Kyoto University, Kyoto, 606-8507, Japan
SOURCE: Journal of Immunology (1999), 163(6), 3007-3011
CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English

AB We investigated the intracellular signaling events of **OX40** ligand (gp34), a member of the TNF family. To elucidate the intracellular signaling via gp34, we prepd. a model system in which a human gp34-transfected mouse epithelial cell line was stimulated with a recombinant sol. form of **OX40**. We demonstrated that **OX40** binding

resulted in increase in c-jun and c-**fos** mRNA levels in this transfectant by Northern blot anal., which was blocked by the pretreatment with anti-gp34 Ab. The studies with various gp34 deletion mutants showed that the cytoplasmic portion including the amino acid sequence 16-21 (RPRFER) was required for the induction of c-jun and c-**fos** mRNA expression. Furthermore, **OX40** binding induced c-jun mRNA expression also in HUVECs, which in our previous study have been shown to express gp34 and interact with activated T cells through the **OX40**/gp34 pathway. On the other hand, c-**fos** mRNA was detectable neither in unstimulated HUVECs nor in gp34-stimulated HUVECs. These results indicate that the **OX40**/gp34 system generates two-way signals and may elicit biol. effects on vascular endothelial cells.

OS.CITING REF COUNT: 35 THERE ARE 35 CAPLUS RECORDS THAT CITE THIS RECORD (35 CITINGS)

REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L35 ANSWER 6 OF 6 BIOSIS COPYRIGHT (c) 2011 The Thomson Corporation on STN

Full Text | Citing References

ACCESSION NUMBER: 2002:632711 BIOSIS
DOCUMENT NUMBER: PREV200200632711
TITLE: Signaling of gp34 (**OX40** ligand) induces vascular endothelial cells to produce a CC chemokine RANTES/CCL5.
AUTHOR(S): Kotani, Ai; Hori, Toshiyuki [Reprint author]; Matsumura, Yumi; Uchiyama, Takashi
CORPORATE SOURCE: Department of Hematology and Oncology, Graduate School of Medicine, Kyoto University, Kyoto, 606-8507, Japan, Japan thori@kuhp.kyoto-u.ac.jp
SOURCE: Immunology Letters, (October 21 2002 2002) Vol. 84, No. 1, pp. 1-7. print.
CODEN: IMLED6. ISSN: 0165-2478.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Dec 2002
Last Updated on STN: 12 Dec 2002

AB We previously showed that gp34 (**OX40** ligand) expressed on vascular endothelial cells is not only involved in adhesion between activated T cells and endothelial cells but also by itself able to transmit intracellular signals leading to expression of c-**fos** and c-jun mRNA upon **OX40** binding. In the present study, we searched for genes that were induced or upregulated by gp34 signaling in human umbilical vein endothelial cells (HUVECs) to define its downstream biological events. HUVECs expressing high levels of gp34 were stimulated with recombinant soluble **OX40** or mock control and subjected to analysis using cDNA expression arrays. We found that a CC chemokine RANTES (regulated upon activation, normal T cell expressed and secreted)/CCL5 is one of such inducible genes. Reverse transcriptase-PCR analysis showed that expression of RANTES mRNA was induced after incubation with soluble **OX40** and this induction was inhibited by anti-gp34 mAb. We could detect expression of intracellular RANTES protein by flow cytometry in HUVECs stimulated with soluble **OX40** as well as fixed **OX40** transfectant cells but not those stimulated with mock supernatants or mock transfectant cells. Again, this induction of RANTES protein was inhibited by anti-gp34 mAb. These results clearly indicate that gp34 signaling induces expression of RANTES at both mRNA and protein levels in HUVECs and suggest a possible link between the **OX40**/gp34 system and RANTES during the process of T cell adhesion to endothelial cells and subsequent extravasation.

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